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VOLUME 16 · NUMBER 3 JULY 1961

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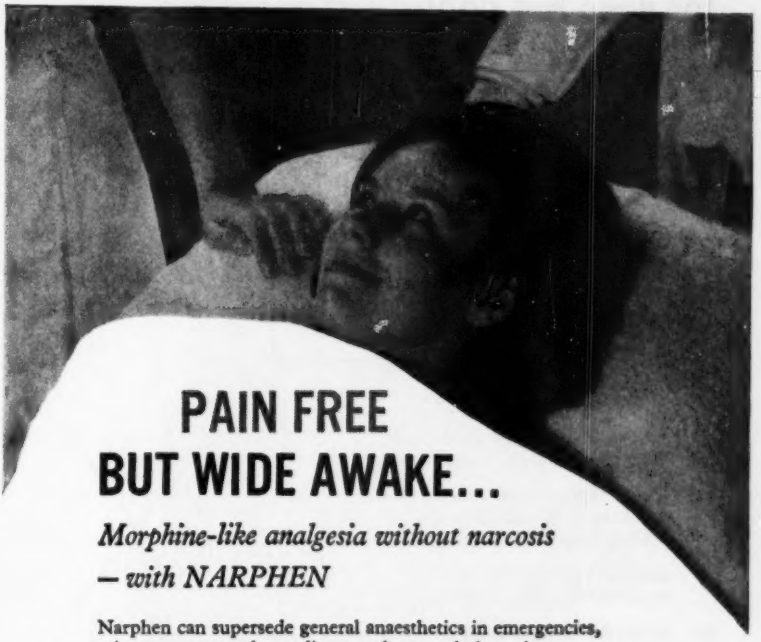
CONTENTS

	Page
EDITORIAL	265
THE TORONTO GENERAL HOSPITAL RESPIRATORY UNIT H. Barrie Fairley	267
THE INTENSIVE CARE UNIT (A three year experience at Baltimore City Hospitals) ... ... P. Safar *et al*	275
CONTROLLED VENTILATION IN MEDICAL PRACTICE O. P. Norlander *et al*	285
EXPERIENCES IN A SMALL RESPIRATORY UNIT OF A GENERAL HOSPITAL (With special reference to the treatment of tetanus) ... ... D. J. Pearce	308
A NEW MECHANICAL VENTILATOR ... ... R. W. Manley	317
CIRCULATORY AND RESPIRATORY EFFECTS OF ETHER, HALOTHANE AND THE AZEOTROPE MIXTURE (A comparative study in children) G. W. Black *and* S. H. S. Love	324
AN INVESTIGATION OF NEW FLUORINE COMPOUNDS IN ANÆSTHESIA (3) (The anæsthetic properties of hexafluorobenzene) T. H. S. Burns *et al*	333
EFFECT OF POSTURE ON VENTILATION OF PATIENTS ANÆSTHETISED WITH HALOTHANE ... ... F. F. Wood-Smith *et al*	340
THE SAFETY OF NEOSTIGMINE ... ... J. E. Riding *and* J. S. Robinson	346
ANATOMY FOR ANÆSTHETISTS (2) (The anterior abdominal wall) H. Ellis *and* M. McLarty	355
THE TOXICITY OF LIGNOCAINE 2 PER CENT A. R. de C. Deacock	363
NEW INVENTIONS	
Attachment for the Ruben valve ... ... C. H. Hoskins	366
Open incubator for neonatal surgery ... ... G. J. Taylor	367
Reception–resuscitation cots for the newborn ... ... R. D. Levis	371
ANÆSTHETIC LITERATURE	374
CORRESPONDENCE	
Hypotension with 'Polybrene' ... S. A. Feldman *and* J. R. Hoyle	382
OBITUARY	
William Stanley Sykes	383
ASSOCIATION NEWS	384
FACULTY NEWS	385
COMMONWEALTH AND FOREIGN NEWS	386



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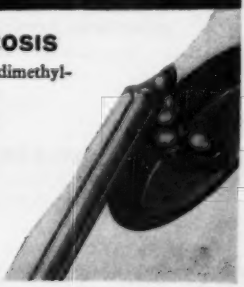

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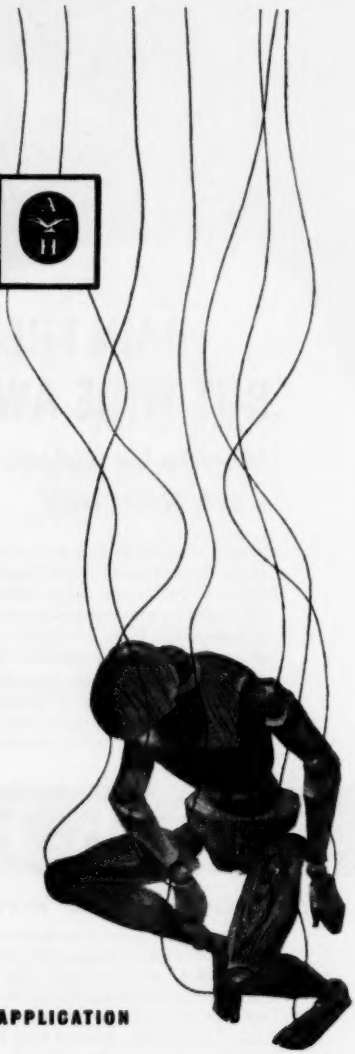

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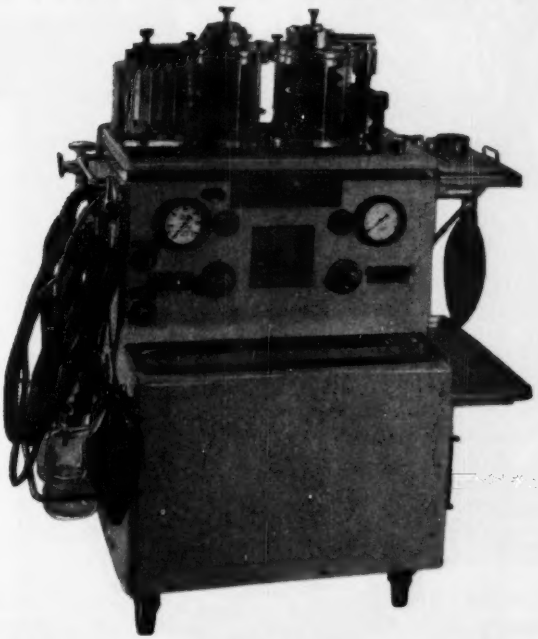

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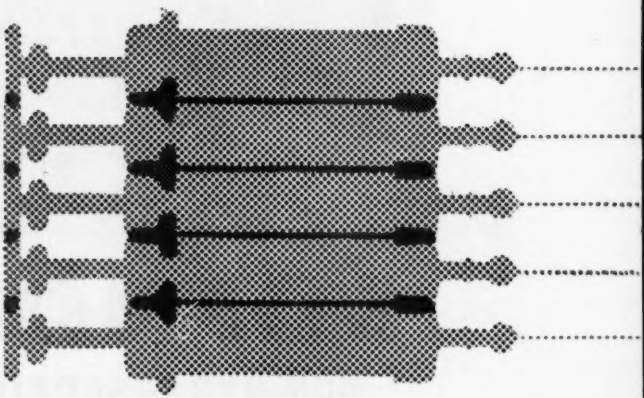

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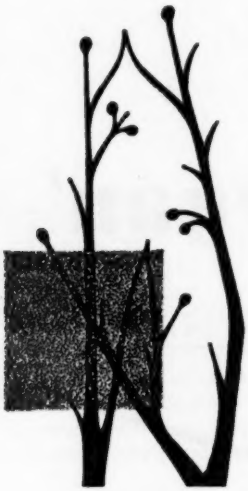

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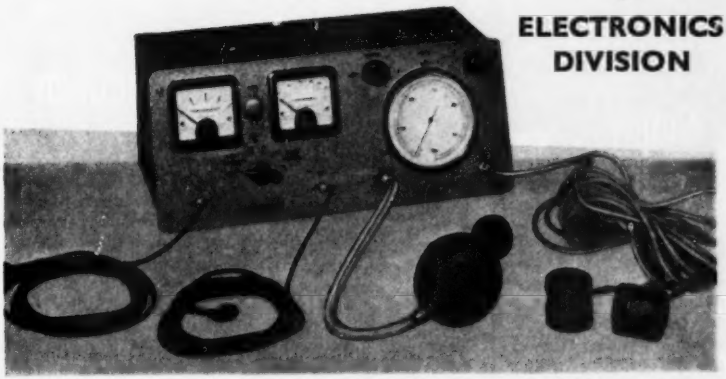

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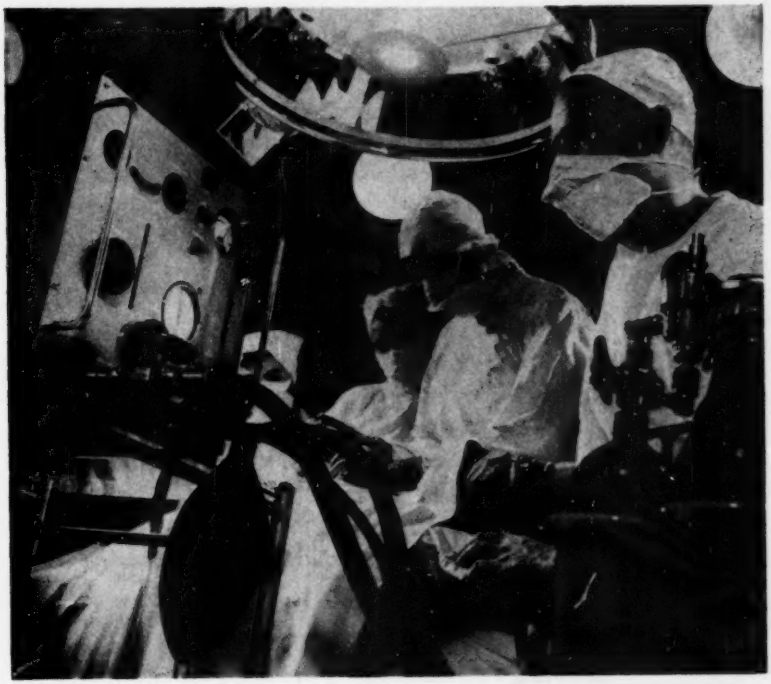

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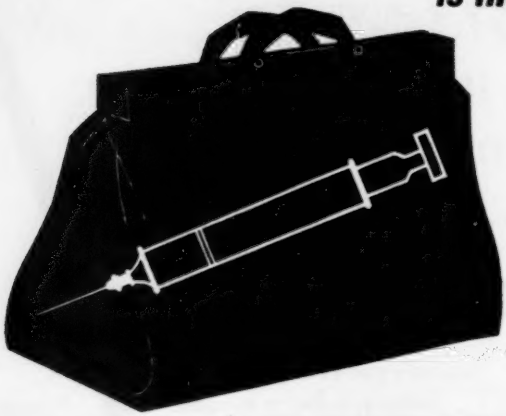

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EDITORIAL

During the past fifteen years the specialty of anæsthesia has been growing up. The training of the anæsthetist has endowed him not only with technical skills, but also with a considerable knowledge of respiratory and circulatory physiology. In addition the modern practice of anæsthesia provides an unrivalled opportunity for exercise in applied pharmacology.

The ability to carry an apnœic patient for long periods in a state of physiological balance has an obvious application in the treatment of acute respiratory failure; and it was in this field that physicians first recognised the value of anæsthetic help. The management of acute respiratory crises arising in so-called bulbar poliomyelitis has become, since the experience in the Copenhagen epidemic of 1953, largely a partnership between physician and anæsthetist. In the last five years this partnership has been extended and now, in many centres, special units have been established for the treatment of diseases in which respiratory insufficiency or failure may be expected or feared. The formation of these units has involved more than the clinical employment of the anæsthetist. The units must be liberally, even lavishly, staffed with highly trained nurses and from this concentration of nursing talent has developed the intensive care unit which deals, of course, with a wider field than respiratory disease. The extensive participation of

pathologists and physiotherapists is also a vital part of the organisation.

At the Second Congress of the World Federation of Societies of Anæsthesiologists in Toronto in the late summer of last year, one afternoon was devoted to discussion of various aspects of the management of acute respiratory disease and of intensive medical and nursing care. We have much pleasure in publishing in this issue papers by four of the speakers who took part in this session in Toronto. The subject is of considerable interest to us as anæsthetists, and although the function of the special unit may vary from one centre to another, the extent of anæsthetic participation is fairly constant. This is true, at any rate, of those units in Toronto, Baltimore, Stockholm and Southampton, from which widely separated centres our four reports come.

In this issue there will also be found a description of a new mechanical ventilator.

RPWS

The Toronto General Hospital Respiratory Unit

H. BARRIE FAIRLEY, MB, BS, FFARCS

Department of Anæsthesia, University of Toronto
and the Toronto General Hospital

At the Toronto General Hospital, prior to October 1958, ventilatory insufficiency was usually treated in the ward on which it was diagnosed. The methods used were various, but the tank and cuirass respirators were most popular. The majority of patients given prolonged respiratory assistance were neurological and the results were poor. Decubitus ulcers, pneumonia and a general inaccessibility of the patient were common.

Recognising the inadequacy of this situation, a special area of the hospital was set aside for the management of respiratory insufficiency, using IPP, and a team of physicians representing the Departments of Anæsthesia, Medicine and Otolaryngology was appointed[1]. This system of a small team of specialists being responsible for such a unit has since proved most successful and in our opinion, greatly superior to control by any one speciality.

A permanent staff of graduate nurses greatly increased the efficiency of the unit, as shown in TABLES 1 and 2:

Table 1

May–October 1958

DIAGNOSIS	TREATED	SURVIVED	DIED
Polyneuritis	2	0	2
Post-operative respiratory failure	7	4	3
Stove-in-chest	3	2	1
Barbiturate overdose	3	3	0
Myasthenia gravis	1	1	0
Disseminated sclerosis	1	1	0
TOTALS	17	11	6

Adapted from a paper originally read at the World Congress of Anæsthesiologists, Toronto, 1960

Table 2

October 1958–March 1959

DIAGNOSIS	TREATED	SURVIVED	DIED
Poliomyelitis	1	1	0
Post-operative respiratory failure	3	3	0
Stove-in-chest	5	4	1
Barbiturate overdose	5	5	0
Myasthenia gravis	1	1	0
Emphysema	4	4	0
Tetanus	1	0	1
Status epilepticus	1	1	0
TOTALS	21	19	2

By coincidence, two very comparable groups of patients were handled by the same medical team in the five months before and the five months after the opening of the unit. The main variable was that the first group was handled in the ordinary wards of the hospital, without specialist nursing care.

Deaths due to such simple causes as airway obstruction occurred in the first group while the fewer deaths in the second group were related to less avoidable complications. The difference in mortality was associated with a parallel difference in morbidity and demonstrates the importance of specialist nurses, in managing such patients.

When the unit was opened, it was anticipated that the majority of patients with respiratory insufficiency would be those suffering from neurological disease. TABLE 3 shows that this proved not to be the case:

Table 3

October 1958–March 1961

DIAGNOSIS	ADMISSIONS	SURVIVED	DIED
Pulmonary disease	54	37	17
Neurological disease	48	34	14
Post-operative respiratory failure	28	17	11
Drug intoxication	33	32	1
Chest injury	17	14	3
Uncompensated metabolic acidosis	7	3	4
TOTALS	187	137	50

In the first twenty-nine months, 187 patients were admitted to the unit, 78 per cent of whom came from other departments of this 1200-bed teaching hospital. The substantial proportion arising from within the hospital itself suggests the frequency of coincidental ventilatory insufficiency in a large busy general hospital.

Patients have been grouped under major headings, for the sake of simplicity. Although this prevents a full appreciation of the wide variety of cases treated, it is evident that a large number of patients were not suffering from neurological disorders.

The largest group of patients – and the most resistant to treatment – were those with severe chronic pulmonary disease with superadded acute respiratory insufficiency. Other cases have been grouped as shown and our experience has been similar to that of other units: *i.e.* patients with paralyses, drug intoxications or crushed chests stand an excellent chance of recovery from their respiratory insufficiency, in the absence of major complicating factors. In this series, typical such factors have been previous cerebral hypoxia, gross hypotension or severe multiple injuries. However, severe pulmonary disease – particularly when associated with air trapping – greatly reduces the chances of survival. Various bizarre conditions are included in the figures. One of these patients was admitted having swallowed caustic and thrown himself into Lake Ontario. Originally diagnosed as a case of drowning with cerebral hypoxia, he was resuscitated only to die two days later of massive gastro-intestinal hæmorrhage, due to the caustic.

TABLES 4, 5 and 6 show the distribution of cases within three of the major groups:

Table 4

Pulmonary disease

DIAGNOSIS	ADMISSIONS	SURVIVED	DIED
Emphysema	43	29	14
Pneumonia	6	4	2
Status asthmaticus	4	4	0
Fibrocystic disease	1	0	1
TOTALS	54	37	17

Of those patients with chronic pulmonary disease, the majority were those with severe emphysema, presenting with marked recent additional ventilatory insufficiency, usually with right heart failure and frequently comatose and *in extremis*. This group proved less rewarding than the others, as one might expect and other methods of treatment – particularly the use of respiratory stimulants – are currently being investigated.

Table 5

Neurological disease

DIAGNOSIS	ADMISSIONS	SURVIVED	DIED
Intracranial aneurysm	1	0	1
Cerebral hypoxia	6	1	5
Head injury	2	1	1
Disseminated lupus E	1	0	1
Disseminated sclerosis	3	3	0
Cervical cord injury	4	3	1
Myelitis	1	1	0
Cerebrovascular accident	1	0	1
Status epilepticus	2	2	0
Myasthenia gravis	11	10	1
Peripheral neuropathy	12	10	2
Poliomyelitis	1	1	0
Tetanus	3	2	1
TOTALS	48	34	14

The types of neurological disorder treated reflect the practice of a university hospital in a city with separate infectious disease facilities. Thus, myasthenia gravis and quadriplegia figure more prominently than poliomyelitis. Survival was the rule in most cases falling in this group. Typical of the extreme complicating factors which occasionally prevented this survival were the sixty-five year old patient with tetanus who, having survived one week beyond the end of a period of very severe spasms, died of septicæmia owing to coincidental Felty's syndrome with a white cell count of 600 per cu.mm and a myasthenic, who died suddenly from secondary carotid artery hæmorrhage ten days after thymectomy and tracheostomy.

Table 6

Post-operative respiratory failure

OPERATION	ADMISSIONS	SURVIVAL	DIED
General surgery	13	8	5
Pulmonary surgery	6	4	2
Cardiac surgery	9	5	4
TOTALS	28	17	11

Cases admitted post-operatively usually presented with a serious complicating factor, such as previous chronic pulmonary disease. The results reflect the severity of this underlying disease, but the survivors suggest the remarkable salvage rate which can be achieved in a group of cases, the majority of whom would be given a minimal chance in many surgical units. Short term cases, treated at Recovery Room level, are not included in TABLE 6. The post-cardiac surgery cases represent a selected group of the worst respiratory problems, treated in the cardiac intensive therapy unit with respirators and transferred to the respiratory unit when difficulties arose in their management.

The concepts arising from three aspects of our experience in this type of work will be discussed: (1) the indications for assisting respiration; (2) the use of muscle relaxants in respiratory insufficiency; (3) the establishment of facilities for the management of ventilatory inadequacy.

Other aspects of the problems associated with the management of respiratory insufficiency are discussed elsewhere[2].

1 INDICATIONS FOR ASSISTING RESPIRATION

There is a variety of circumstances where respiration should be assisted. These may be grouped under four headings:

(1) Where frank underventilation is demonstrated by clinical assessment, ventilatory volume measurement and arterial carbon dioxide levels. This has been the commonest indication for assisted ventilation, in our experience. Clinical assessment has been found relatively un-

satisfactory. Consequently, with the exception of the extreme emergency, ventilatory volumes are measured with a Wright Anemometer and compared with the normal values shown by the Radford nomogram. The possibility of an enlarged physiological dead space altering this suggested normal value is considered. If these measurements suggest the possibility of underventilation, confirmation is obtained by arterial puncture and determination of pH and pCO_2. Facilities for these estimations are available on a twenty-four hour basis. In relation to these arterial values, it is our practice to determine pH with carbon dioxide and to assess overall acid-base status in terms of the pH-bicarbonate diagram. In this way, the required pCO_2 can be estimated relative to the metabolic status. Working on this basis, many patients with marked metabolic acidosis may be considered to be underventilating, despite a normal or low pCO_2. Inadequate respiratory compensation of this type has been seen in such combinations as renal failure plus pneumonia.

(2) Those patients in whom the breathing reserve is minimal. Adequate ventilation is only being achieved by maximum effort which will inevitably lead to exhaustion and in many instances, sudden death. Two types of patient fall into this category. One is the neurological patient with muscular paralysis, who is followed daily or twice-daily with spirograms until the vital capacity approaches the tidal volume. The other is the patient with pulmonary disease – frequently accentuated post-operatively – in whom the impending exhaustion is more obvious without measurement. Patients who have undergone cardiac surgery are a special example of this problem.

(3) Stove-in-chest with paradox responds dramatically to IPP and to date, no surgical stabilizing procedures have been considered necessary since the opening of the respiratory unit in this hospital.

(4) Certain patients develop respiratory insufficiency as a result of their therapy. Examples of this are those given muscle relaxants in the treatment of tetanus and status epilepticus.

2 MUSCLE RELAXANTS

Muscle relaxants have been used frequently in the initial management of these patients: an endotracheal tube is passed as soon as respiratory insufficiency is diagnosed and a short-acting relaxant is used at this time, in unconscious patients. Attempts to omit this resulted in two instances of vocal cord granulomata in the present series. In one, with drug intoxication, the tube was only in place eight hours, but, in both, the intubation was difficult and it is felt that trauma at intubation was the causative factor.

Again in the initial management, one dose of relaxant has been given

occasionally to establish a patient on a respirator. This has been especially useful in stove-in-chest with tachypnœa, for example.

Relaxants have also proved of value in nineteen cases, at times other than the initial management, as shown in TABLE 7. (The word 'benefited' indicates survival beyond the point where relaxants were discontinued and the initial indication was corrected.)

Table 7

Prolonged use of relaxants

INDICATION	CASES	BENEFITED
Control spasms	5	5
Control restlessness	2	2
Reduce resistance	6	1
Control tachypnœa	6	2
TOTALS	19	10

Muscle relaxants have successfully terminated convulsions in tetanus and status epilepticus on the five occasions they have been used. On two occasions they were successful in controlling a marked stage of excitement in patients rousing from barbiturate poisoning, where further sedation seemed illogical.

They have been used with effect in the treatment of severe pulmonary disease in two circumstances: (1) where tachypnœa could not be corrected despite high doses of narcotics; (2) where resistance to intermittent positive pressure was such that the necessary inflation pressure produced cardio-vascular embarrassment. Although relaxants have usually achieved their aim in such circumstances and have reduced the necessary pressure, the circumstances rendering them necessary carry a grave prognosis, as shown by the low recovery rate.

In long term relaxant therapy, laudexium is preferred. A dose of 30–60mg is given intravenously at the outset, followed by 6–12mg IM every hour, with adjustments as indicated. No complications have been observed.

The completely relaxed patient is treated in the same way as those with paralysing neurological diseases, passive limb movements, chest physiotherapy and eye care being carried out frequently.

Tranquilisers are given in small doses and care is taken to guard conversation in front of these alert, but apparently comatose patients. In practice, minimal muscular response from the patient should be maintained. He can then indicate awareness and level of consciousness remains as a most important sign. To this end, it is considered inadvisable to combine heavy doses of central depressants with relaxant

therapy in conditions where these may be alternative forms of treatment.

3 FACILITIES FOR MANAGING RESPIRATORY INSUFFICIENCY

Emphasis has been laid on the wide variety of illnesses, all of which are serious, which may be complicated by respiratory insufficiency. The importance of adequate numbers of specialist nurses has been mentioned.

Our experience has been that, in these days of an overall nursing shortage, many seriously ill patients are brought to our attention as possible candidates for the Respiratory Unit, on the grounds that they may well develop respiratory insufficiency and that the intensive nursing care available in the unit may avoid this. (The Toronto General Hospital currently does not have an intensive therapy unit open to all services.) Bed shortage usually prevents our accepting such latent respiratory problems, but there is no doubt that, with good intensive nursing care, respiratory insufficiency may be circumvented in many instances by meticulous attention to such details as control of the airway, of secretions and of fluid balance.

It would seem logical, therefore, not to draw a sharp line between the seriously ill patient requiring intensive care, who may develop bronchopneumonia, secretional obstruction and respiratory insufficiency, and the patient who has actually developed the latter. Consequently, one might suggest that the large general hospital, handling a heavy volume of seriously ill patients, but chronically short of nurses, should have an intensive care unit with an adjacent area specialising in the treatment of respiratory insufficiency.

Consideration should also be given to the advisability of organising facilities for such treatment in circumstances other than the larger centres. In this connection the following factors are relevant: (1) respiratory insufficiency will be an infrequent event in anything other than the larger hospitals; (2) specialist nurses and medical staff, with clinical acumen maintained by constant experience, are essential for maximum success; (3) transportation of a patient with respiratory insufficiency is not difficult, in the absence of gross cardio-vascular instability; (4) some cases of respiratory insufficiency will be of short duration, in particular poisoning with short-acting barbiturates.

With this in mind, anæsthetists should be acquainted with the principles of this type of work during their training. Any hospital busy enough to justify specialist anæsthetist services could be equipped with an automatic ventilator and would handle short-term respiratory insufficiency problems. However, long-term problems might best be

served by transporting them to a unit staffed by specialist nurses and thus be given the improved prognosis which such attention offers.

Acknowledgements

The author wishes to emphasise the contribution of his colleagues on the TGH Respiratory Unit staff: Drs H. O. Barber, R. A. Chambers and C. R. Woolf.

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The Intensive Care Unit

A three year experience at Baltimore City Hospitals

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In September 1958, the Department of Anesthesiology of the Baltimore City Hospitals in combination with the other clinical services and the nursing staff established an Intensive Care Unit[1]. Initially this was an independent unit, but six months after its establishment and due primarily to a desire to utilise the available personnel and equipment most advantageously, it was combined with the existing post-anæsthesia recovery room into a single unit. The purpose of this combined unit was to provide optimal medical and nursing care to all critically ill patients. Both medical and surgical patients were to be admitted to this unit to obtain the benefit of continuous, specialised attention and treatment. Anæsthesia residents and staff anæsthesiologists, who have acted as consultants in the treatment of all respiratory emergencies and of all comatose patients, have found that the combined unit greatly facilitated their work because of concentration of patients and equipment in one area.

This paper is written in the hope that some of our experiences with such a unit may be of interest to our colleagues.

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(A) PROGRESSIVE PATIENT CARE

The establishment of the Intensive Care Unit at Baltimore City Hospitals was the final step in the accomplishment of what is called 'progressive patient care'. The concept of 'progressive patient care', pioneered by the Manchester Memorial Hospital in Manchester, Connecticut, organises the hospital facilities and staff around the intensity of care which patients require rather than, in the conventional fashion, depending upon the patient's disease[2,3]. Accordingly the progressive patient care programme is divided into five phases. (1) Intensive Care Unit; (2) Intermediate Care Unit or Acute Unit; (3) Chronic Care Unit; (4) Self Care Unit; and (5) Home Care Unit. Among the larger teaching hospitals the 2000 bed municipal Baltimore City Hospitals has been operating a four phase programme since 1936. All five phases did not become effective until the establishment of the Intensive Care Unit.

(B) ECONOMICS

The approximate average cost per patient day at the Baltimore City Hospitals is shown in TABLE 1. It is apparent from this table that com-

Table 1

Progressive patient care at the Baltimore City Hospitals, 1960

	BEDS	AVG. COST PER PATIENT/DAY (APPROX.)
Intensive Care Unit-Recovery Room	17–20	$50–80
Acute Unit	375	$27.00
Chronic Unit	431	$13.00
Tuberculosis Unit	337	$ 9.00
Infirmary and Self Care Unit	920	$ 4.00
AVERAGE TOTAL	2083	$25.00 including physician's services

plete progressive patient care organisation tends to lower overall hospital operating costs while improving the care of the critically ill patient. The addition of an Intensive Care Unit in a conventional hospital, not operating on the progressive care principle, results in heavy initial expenditures for equipment and for structural changes. Indeed in private hospitals the high patient day cost of the Intensive Care Unit must be weighed against the added safety and more effective therapy to the critically ill patient. In only one area will the Intensive Care Unit lower the cost of hospitalisation to the critically ill patient and that is the elimination of special duty nurses which under the prevailing fees in this country may become an almost unsupportable burden to many patients.

(C) ORGANISATION

The Intensive Care-Recovery Room Unit at Baltimore City Hospitals consists of two eight-bed wards separated from each other by a low partition. One of these wards is set aside for patients recovering from general or spinal anæsthesia while the other ward is reserved for patients remaining in the Unit for several days or weeks (FIG. 1). Two

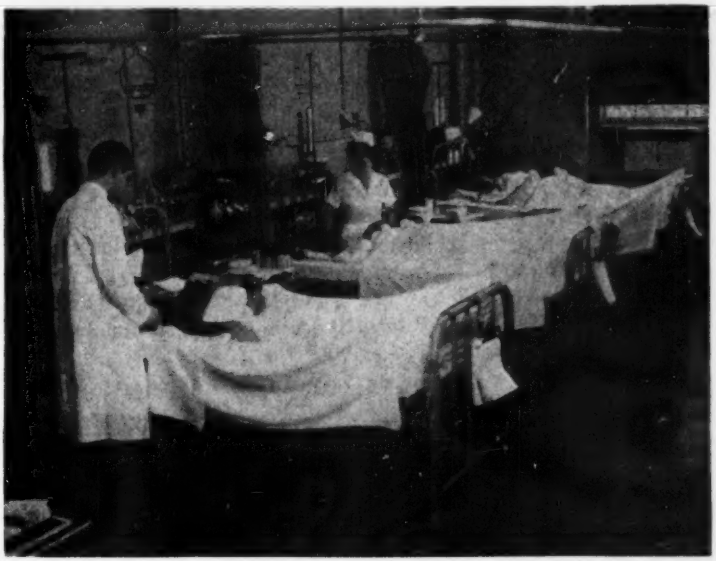

FIG. 1 View of intensive care-recovery unit. These three patients are totally paralysed and are given artificial respiration by the intermittent positive pressure method described in the text

side rooms provide two beds each for isolation of patients suffering from infectious diseases and for the benefit of patients whose conditions require quiet or darkened surroundings. We realise that ideally the post-anæsthetic recovery room should be separate and located on the operating room floor while the independent Intensive Care Unit should be located as near by as possible. Unfortunately due to limitations in funds, physical facilities, and a severe shortage of nurses we had to combine the post-anæsthetic recovery room with the Intensive Care Unit in the above described manner.

It was not necessary to place the unit under the direction of a single department or any one physician. The primary responsibility for any patient admitted to the unit rests with the physician or surgeon admitting the patient. The necessary policies for the unit were established jointly by the Chiefs of Anæsthesiology, Medicine, Obstetrics and Gynæcology and Surgery, the director of the nursing service and the hospital director. The anæsthesiologists act only as consultants although they may occasionally be requested to assume the temporary management of a patient.

The department of anæsthesiology is charged with maintaining the continuity of the various special treatments in the unit, with coordinating the routines of the unit and specifically, with resuscitation and respirator-care. It is also our responsibility to teach resuscitation, respirator-care, tracheotomy care and other routines to the house officers of all services and to the nursing staff.

A separate small Intensive Care-Recovery Area has been established on the pediatric floor because of the special problems involved in the care of infants and small children. This presents a special situation and should certainly not be followed for other clinical services since such multiple units would defeat the main purpose of Intensive Care concept, namely the concentration of personnel and equipment.

(D) ADMISSIONS AND DISCHARGES

The senior house officer on each clinical service is responsible for admitting and discharging of patients of his service to the Intensive Care Unit. The staff anæsthesiologist has to assign priority to patients to be admitted on the basis of the most critical needs. Due to limitations of bed space in the unit it was found to be most important to guard against the admission of non-salvageable patients. It is equally important to guard against the tendency of house officers to keep patients in the unit far beyond the time of true need for intensive care. Discharges from the unit are usually done following a discussion between the staff anæsthesiologist and the physician responsible for the patient.

(E) NURSING

The combined Intensive Care-Recovery Room Unit is established as a separate nursing entity and staffed by registered and trained practical nurses who work permanently in this area. Soon after the establishment of the unit we discovered that nursing in this area required special skills not ordinarily available in the average hospital. This situation was remedied by conducting a special course for all Intensive Care-Recovery Room nurses (TABLE 2). Each lecture in this course was followed by a practical demonstration, and wherever possible, by supervised practice

Table 2

Course for I.C.U. nurses

		INSTRUCTOR
1	Recognition and treatment of respiratory distress, determination of blood pressure, pulse, respiration, respiratory and circulatory resuscitation	Anæsthesiologist
2	Oxygen therapy	Anæsthesiologist
3	Tracheotomy	Anæsthesiologist
4	Respirators	Anæsthesiologist
5	Respirators	Anæsthesiologist
6	Hypothermia	Anæsthesiologist
7	Spinal anæsthesia	Anæsthesiologist
8	Post-operative pain	Anæsthesiologist
9	Care of unconscious patient	Anæsthesiologist
10	Maintenance of patient's chart	Surgeon
11	Shock and I.V. therapy	Surgeon
12	Determination of blood pressure, pulse, respiration	Surgeon
13	Patient after gastro-intestinal surgery	Surgeon
14	Patient after chest surgery	Surgeon
15	Patient after cardio-vascular surgery	Surgeon
16	Patient after orthopædic surgery	Surgeon
17	Patient after urological surgery	Surgeon
18	Patient after neurosurgery	Surgeon
19	Fluid and electrolyte therapy	Surgeon and Internist
20	Tuberculosis, isolation	Internist
21	Physical therapy	Internist
22	Special problems in care of medical patients	Internist
23	Special problems in care of obstetrical and gyn. patients	O.B.–Gynæcologist
24	Summary of techniques and equipment	Anæsthesiologist
25	Exam	Anæsthesiologist

on patients in the unit. The course proved very successful and several nurses working in other parts of the hospital were included among the students so that a reserve of trained personnel be available in case of illness among the staff or in case of a sudden influx of critically ill patients to the unit.

On rare occasions, during the initial period of our experiences in critical patient care, special duty nurses from an outside registry were used. This approach proved to be entirely unsatisfactory since these nurses, although adequate for ward care, proved to be unsuitable for intensive care in the Unit, being unfamiliar with the special techniques and the routines required.

(F) EQUIPMENT

The equipment in the Intensive Care-Recovery Unit should be as simple as possible and techniques should be standardised so that they will be well understood by all the personnel involved. The resuscitation equipment, respirators, oxygen equipment and nebulisers, are concentrated in the unit and are maintained by an oxygen therapist in co-operation with the Intensive Care-Recovery Unit nursing staff. It is our impression that loss and breakage of equipment is reduced because only trained people handle this special equipment. The Intensive Care-

Recovery Unit is air-conditioned and thus we find little if any use for oxygen tents which hamper nursing care, make it more difficult to observe patients and usually provide lower effective concentrations of oxygen than the face tent or tracheotomy mask.

(G) TREATMENT

The conditions treated in the Intensive Care-Recovery Unit during the twenty-four months of 1959–61 are shown in TABLE 3.

Table 3

Patients admitted to I.C.U./Recovery Room in two years*

	NO ADMITTED	NO DIED
SURGERY/GYN.–OBST.		
Post-operative without complications	3370	0
Post-operative with complications	224	55
Head injury	57	14
Severe burns	5	4
Severe chest injury	8	1
TOTAL	3664	74
MEDICINE		
Pulmonary œdema	44	19
Pneumonia	39	19
Coronary occlusion	32	14
Poisoning and coma	30	3
Cerebro-vascular accident	20	13
Convulsions	13	3
Septicemia	10	5
Obstructive emphysema	7	1
Respiratory paralysis (polio, etc)	18	2
Others	54	17
TOTAL	267	96

*March 1959/61

Since our Intensive Care Unit also functions as a polio respirator centre for the City of Baltimore and surrounding areas it seems appropriate at this point to digress somewhat on our approach to chronic respirator care.

For controlled respiration (intermittent positive pressure artificial respiration) we have been using up to eight Moerch piston respirators at the same time. This respirator proved reliable for long-term use and is simple to operate (FIG. 2). During the poliomyelitis epidemic in the autumn of 1960 about twenty-five patients (as many as thirteen patients simultaneously) were ventilated with this type of equipment. Because of the large stroke provided by this respirator it was possible, in an emergency, to ventilate two adult patients simultaneously with one respirator. The tidal volumes provided for each patient could be adjusted somewhat by changing the diameters of the tubings.

FIG. 2
The Moerch piston respirator in use

The patients requiring prolonged artificial respiration are tracheotomised immediately upon admission and a cuffed tracheotomy tube is inserted. The Moerch modification of the Jackson Silver Cannula, with swivel attachment, is most satisfactory (FIG. 3). The cuff is inflated but not completely, permitting the escape of some air around the cuff, thus enabling the patient to speak and yet provide a fairly constant amount of ventilation. In patients in whom aspiration is likely to occur due to absence of laryngeal reflexes, the cuff remains completely inflated, just sufficiently to abolish leakage; tracheal necrosis is prevented by deflating the cuff briefly every two to three hours. The tracheotomy tube is changed daily after the first forty-eight hours and suctioned whenever necessary, the most rigid aseptic technique being maintained during suctioning at all times. No suction catheter is re-used without first being sterilised and no suction catheter is used for tracheal and pharyngeal suctioning at the same time.

FIG. 3 View of the Moerch modification of the Jackson tracheotomy cannula. The non-rebreathing valve of the respirator, the manometer to monitor intratracheal pressure, and the Wright ventilation meter are also shown

Artificial respiration is provided at a rate and tidal volume of about one and one-half to two times that recommended by the Radford nomogram. This usually prevented 'air hunger' in the conscious patient and produced mild respiratory alkalosis. FIG. 3 shows how tidal volumes and inflation pressures are monitored during use of the piston respirator. For checks at intervals a Wright ventilation meter is placed between the non-rebreathing valve and tracheotomy tube, measuring exhaled tidal volumes. With this constant volume ventilation the airway pressure, which can be maintained continuously, rises when the pulmonary resistance increases (*e.g.* mucus plug in bronchus).

If, in spite of respiratory alkalosis, there is cyanosis or oxygen desaturation in an arterial sample (arterial samples were taken only occasionally) the oxygen concentration in the respirator air is increased. This often is necessary in patients with severe pulmonary disease.

With the régime outlined routine blood gas analyses were found to be unnecessary. The indirect estimation of the arterial carbon dioxide tension by the rebreathing method of Collier and Campbell[4] was found practical also for use on apnœic patients and seems helpful in monitoring ventilation routinely without the need for arterial puncture.

Whenever the patient's own respiratory efforts interfered with the

rhythm of the respirator, small amounts of muscle relaxants or narcotics were administered. These may have to be repeated a few times, although usually the patient once rendered alkalotic, tolerates the respirator remarkably well.

One of the major problems with the old style piston respirator was the difficulty in humidifying the air. This difficulty was overcome

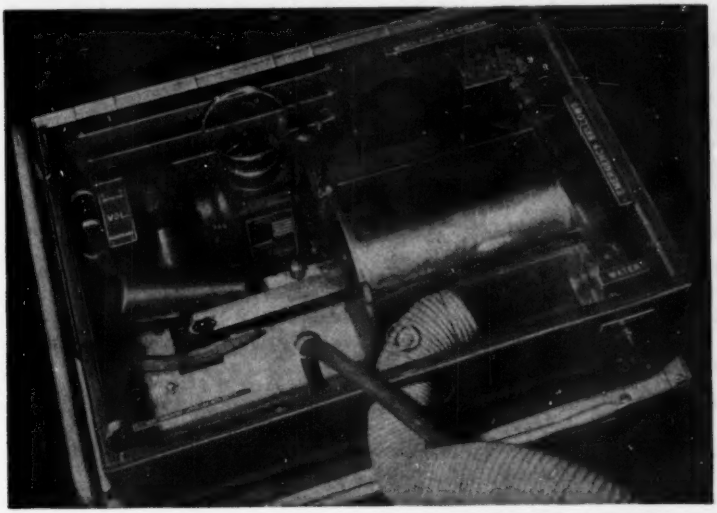

FIG. 4 The large-bore corrugated delivery tube of the Walton humidifier is shown, placed into the intake port of the Moerch respirator

initially by connecting a Walton type humidifier to the intake port of the respirator (FIG. 4) and later by different types of improvised heated humidifiers, placed between the piston and the non-rebreathing valve. These heated humidifiers provided a relative humidity in the inspired air of over 90 per cent.

For assisted respiration and for controlled respiration of conscious patients who are not tracheotomised and who can be ventilated via a mouth-piece, pressure cycled respirators of the Bennet and Bird type are also available. The main use of these respirators was found in patients suffering from rapidly reversible conditions, such as acute pulmonary œdema and status asthmaticus.

It is essential that patients on long-term artificial respiration be watched extremely closely and for this reason one of the anæsthesia residents is assigned on a full-time basis to the Intensive Care-Recovery

Unit. During the height of the poliomyelitis epidemic two anæsthesiology residents and two medical house officers were assigned to the Unit at all times.

(H) THE ROLE OF THE ANÆSTHESIOLOGIST

About 30 per cent of our residents' time is spent in answering requests for consultations and providing emergency resuscitation.

The primary function of the anæsthesiologist in the treatment of patients in the Intensive Care-Recovery Unit is to supplement and coordinate the activities of the other specialists. By providing expert resuscitation of the seriously ill patient and by providing adequate ventilation and support of circulation, the anæsthesiologist can give the internist or surgeon enough time for diagnosis and treatment of the underlying disease process.

Acknowledgements

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Controlled ventilation in medical practice

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During the extensive epidemics of poliomyelitis in Scandinavia 1950–3 it was soon found out that controlled ventilation achieved by intermittent positive pressure ventilation could save a large number of

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patients hitherto having a high rate of mortality[8,14,15]. Great experience was gained with this kind of treatment especially regarding the performance and the physiological effects of different types of mechanical ventilators.

With this knowledge it was apparent that the principles for controlled ventilation should be applicable also to patients after major thoracic surgery with ventilatory insufficiency. The successful results of this treatment with the Engström Respirator were first published in 1955 and 1957[2,3,5]. From that time the method of prolonged controlled ventilation after surgical or accidental trauma as described by Björk and Engström has been widely accepted at most Swedish hospitals. The method has also found a wider application for other indications and diseases where the respiratory system has been pathologically changed, as it was evident that the application of ventilation with the Engström Respirator *per se* could be regarded as involving very little risk to the patients.

INDICATIONS AND PRINCIPLES

Ventilatory insufficiency

Alveolar hypoventilation results in carbon dioxide retention and inadequate oxygenation. Due to increasing levels of carbon dioxide the pH will decrease, *i.e.* respiratory acidosis. Inadequate oxygenation sooner or later results in metabolic acidosis with further demands on the elimination of carbon dioxide. With an already existing alveolar hypoventilation the respiratory acidosis may be even more pronounced. It is also evident that severe metabolic acidosis in itself may be the cause of a relative hypoventilation, *i.e.* the respiratory response of the patient is not enough to compensate for increasing levels of fixed acids. Acidosis and hypoxia will lead to impaired circulation. If that condition is not treated in time, the patient will succumb[11].

The underlying cause of these disorders is insufficient respiratory work of the patient. A difference between the respiratory work needed for adequate ventilation and the respiratory work the patient is able to perform will result in respiratory anoxæmia and acidosis. A respiratory insufficiency thus may have the following causes: (1) A decreased ability to perform respiratory work, for example, in poliomyelitis, intoxications, cerebral trauma, myasthenia gravis, tetanus and in conditions of general shock. (2) An increased demand of respiratory work due to unfavourable conditions for ventilation, such as massive pulmonary infiltrations, pulmonary fibrosis, 'fluid lung', pulmonary œdema, pulmonary hypertension, emphysema, bronchial and tracheal

stenosis, status asthmaticus, thickened pleura with fixation of ribs and diaphragm, multiple rib fractures, distended abdomen with the diaphragm locked in expiratory position (ileus), tracheo-bronchitis, mediastinal emphysema and metabolic acidosis (renal insufficiency).

A combination of these different factors is often found in many patients and it is sometimes difficult to diagnose the primary cause.

Whatever the causative factor of respiratory insufficiency may be, there is an indication to increase the alveolar ventilation by tracheostomy which will decrease the dead space and thus improve the alveolar part of the total ventilation and also facilitate the removal of secretions by aspiration. If tracheostomy alone is not sufficient to improve the condition of the patient to a considerable degree, controlled ventilation should be applied. Controlled ventilation will entirely relieve the patient of the respiratory work and ensure an adequate ventilation in spite of unfavourable mechanical conditions.

TECHNIQUE

Tracheostomy

A tracheostomy should be made as early as possible, as soon it is evident that the patient might develop respiratory complications that may lead to hypoventilation. The tracheostomy should be performed during quiet conditions, if possible under a guarantee that the patient has an open airway without any obstruction. It is often advisable to perform the tracheostomy in direct sequence after the surgical intervention, if it has been decided that the patient might need further ventilatory treatment. An endotracheal tube or a bronchoscope ensures a free airway. This is especially important when tracheostomies are made for upper airway obstruction in small children. The tracheostomy should be placed as high as possible (second to fourth tracheal cartilages) otherwise it is difficult to obtain an airtight connection using an ordinary cannula with an inflatable rubber cuff. The rubber-cuffed, double silver cannula is preferred to a cuffed rubber tube which may be dislocated down into the right main bronchus. Furthermore a rubber tube may cause pressure necrosis. With the double silver cannula such pressure necrosis has not been observed in any of our patients. The double cannula also facilitates cleaning and control of the airway and gives a maximal lumen (FIG. 1). The widest possible cannula should be used. In small children a single silver cannula without a cuff may be used in order to ensure maximal width of the airway. Airtight connection is achieved by pressing the cannula into the trachea of the child.

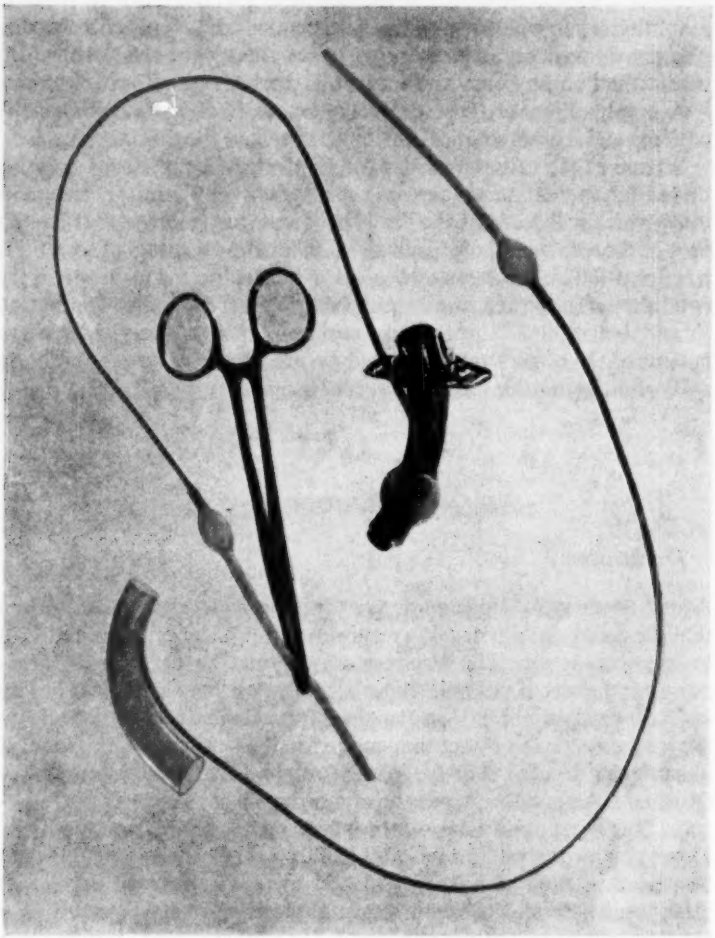


FIG. 1 The double lumen silver cannula with the specially moulded rubber cuff

THE RESPIRATOR AND GAS KINETICS OF CONTROLLED VENTILATION

The controlled ventilation has been administered in all patients by a special device presented by Engström 1951 (FIG. 2). In recent years the apparatus has come into widespread use for the treatment of severe cases of respiratory malfunction.

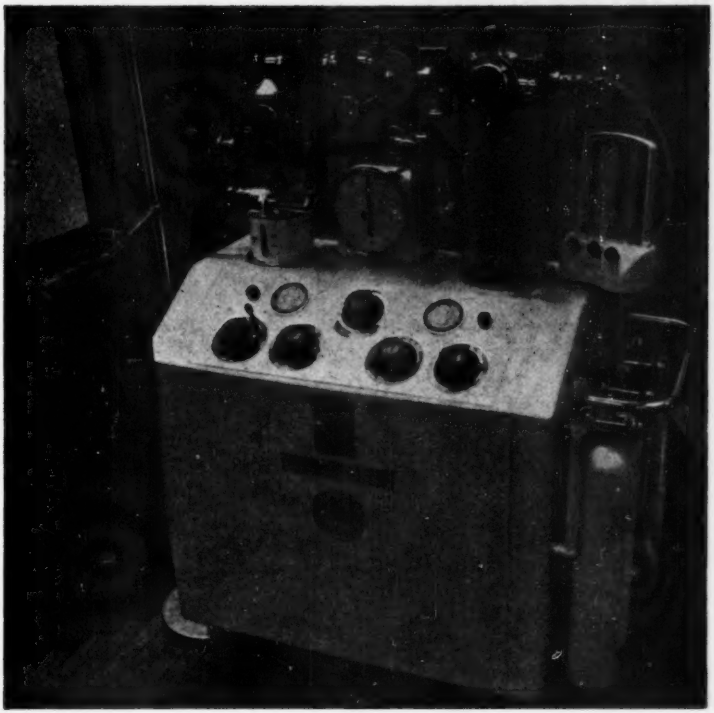


FIG. 2 The Engström Respirator Model 200. The apparatus can be used either in a non-rebreathing system or in a closed or semi-closed system for anæsthesia purposes

Principally the apparatus operates by direct insufflation of a predetermined tidal volume into the patient's airtight air passages. For post-operative use the apparatus is working with a non-rebreathing system. However, for anæsthetic purposes the apparatus can also be used in a closed or semi-closed system.

The apparatus generates a flow through the action of a piston in a cylinder which in its turn acts on a insufflation balloon connected to the patient system. The apparatus, therefore, consists of *two* operating circuits – the circuit connected to the patient which includes the insufflation balloon, and the circuit which delivers the power to compress and fill the insufflation balloon with the predetermined tidal volume (FIG. 3). The power part consists of a piston in a cylinder able to deliver flow rates up to 360 l/min at pressures of more than 70cm

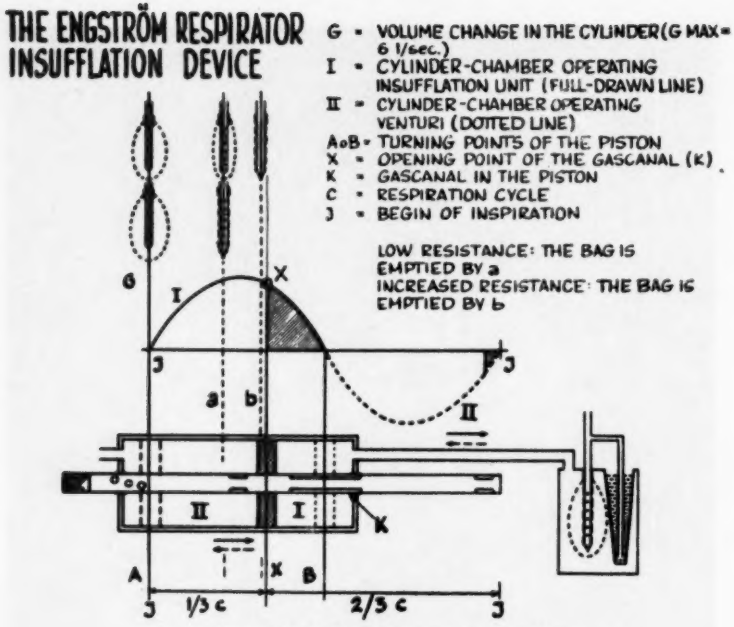


FIG. 3 Power device of the Engström Respirator

water. The piston moves according to a sine shaped curve back and forth. Insufflation corresponds to $\frac{1}{3}$ of a complete breathing cycle or $\frac{2}{3}$ of the stroke during the compression phase. During this phase the insufflation bag is compressed and completely emptied. When the piston moves in the opposite direction a vacuum is now created in the plexiglass cylinder which surrounds the insufflation balloon which is filled up with a preset volume of gas, either room air or a mixture of room air and oxygen – (during anæsthesia, the anæsthetic mixture) through a graduated inlet valve or through rotameters. At the same time a strong positive pressure is created in the second half of the cylinder which pressure creates a negative suction through a venturi on the patient's circuit when three-tenths of the expiratory period has elapsed.

The accuracy of preset tidal volumes amounts to $\pm$ 5ml which is of importance when the respirator is used for controlled ventilation of newborns. The mechanical dead space as well as the compressible volume of the apparatus is exactly known. Therefore, an exact amount of alveolar ventilation can be delivered to the patient.

The tidal volume is administered with the following flow-pressure pattern (FIG. 4): the flow starts from zero and accelerates to a peak

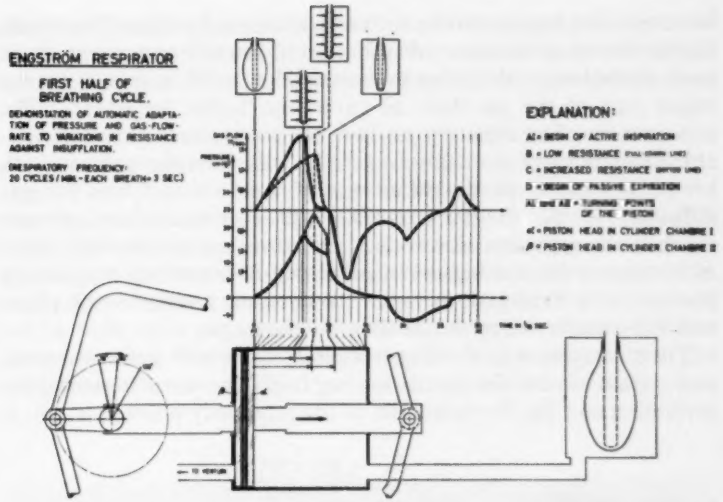

ENGSTRÖM RESPIRATOR
FIRST HALF OF BREATHING CYCLE.
DEMONSTRATION OF AUTOMATIC ADAPTATION OF PRESSURE AND GAS-FLOW-RATE TO VARIATIONS IN RESISTANCE AGAINST INSUFFLATION.
(RESPIRATORY FREQUENCY: 20 CYCLES / MIN. = EACH BREATH = 3 SEC.)
EXPLANATION:
A = BEGIN OF ACTIVE INSPIRATION
B = LOW RESISTANCE
C = INCREASED RESISTANCE
D = BEGIN OF PASSIVE EXPIRATION
A1 and A2 = TURNING POINTS OF THE PISTON
α = PISTON HEAD IN CYLINDER CHAMBER I
β = PISTON HEAD IN CYLINDER CHAMBER II

FIG. 4 Power device and first half of breathing cycle

velocity which can be as high as 6 l/sec. The peak velocity of the gas flow is dependent on the effective resistance (impedance) of the patient's air-containing system. When the tidal volume in the insufflation bag is completely emptied, the flow velocity will drop to zero again. This period corresponds to the dynamic phase and can, in patients with normal impedance, be completed in sixth-tenths of the inspiratory time period. During the remaining part of the inspiratory period the insufflation bag is still compressed by the pressure built up by the piston in the cylinder and a static phase of inspiration is created. The dynamic and static phases correspond to one-third of a complete breathing cycle or to two-thirds of the way the piston travels during compression in one direction. At that point a pressure release is created in the cylinder. The insufflation balloon will, therefore, be at atmospheric pressure and the pneumatic expiration valve can open, which permits the passive expiration of the patient. Due to this arrangement the relationship between dynamic and static inspiratory phases will always adjust itself to what is most suitable for the patient. During the period of early, slow gas flow, gas will pass through even partially stenosed or constricted bronchi. This slow gas flow in these parts corresponds to an increase in volume distal to the stenosis and a concomitant pressure rise will occur which will further widen the bronchi. Such patients will, therefore, be able to receive gas flow of higher

velocity and a larger volume without increasing frictional resistance. During the static phase a redistribution of gas will occur from those parts of the lungs, which due to low impedance will have received the major part of the gas flow, to parts with higher impedance. The arrangement of accelerating gas flow and static phase permits a suitable distribution of gas at lowest possible mean alveolar pressure with lowest amount of pendulum air and optimal contact time for gas diffusion over the alveolar membranes. This favourable arrangement cannot be brought out in any other respiratory system. All other types of ventilators have either non-accelerating flow and too long acting plateaus or a fixed ratio between the dynamic and the static phase which are unaffected by the condition of the lungs.

The arrangement in the Engström Respirator with automatic pressure release around the insufflation bag is of importance regarding the expiration and the characteristics of the expiratory phase (FIG. 5). A

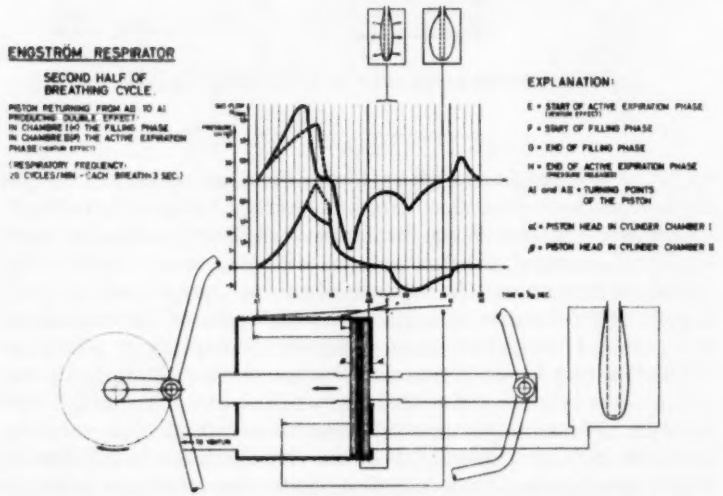


FIG. 5 Power device and second half of breathing cycle with the action of the venturi

period of passive expiration precedes the negative phase. This passive period avoids bronchial collapse and trapping of gas in the lungs, which otherwise will occur if the negative phase is directly applied at the start of expiration. Gentle application of suction is required to avoid bronchial collapse and gas trapping even after a period of passive expiration. The venturi system in the Engström respirator makes it possible to evacuate a large volume in short time at a low negative

pressure. The flow pattern of the venturi system corresponds to that of the inspiratory flow and is activated by the piston in the cylinder in the opposite cylinder chamber. The ratio between inspiration to expiration is 1:2.

Spontaneous respiration of the patient does not influence the action of the respirator. The insufflation balloon is under atmospheric pressure except for the very short periods of insufflation and filling. As soon as the balloon is filled, which occurs before the expiration is finished, the patient may take a breath at atmospheric pressure from the balloon. The next tidal volume will be that amount less than that the patient inhaled spontaneously – thus no dangerous overpressures can be built up in the system. The minute ventilation stays also unchanged, which facilitates synchronisation of the patient with the respirator without resorting to muscle relaxants (FIGS. 6 and 7).

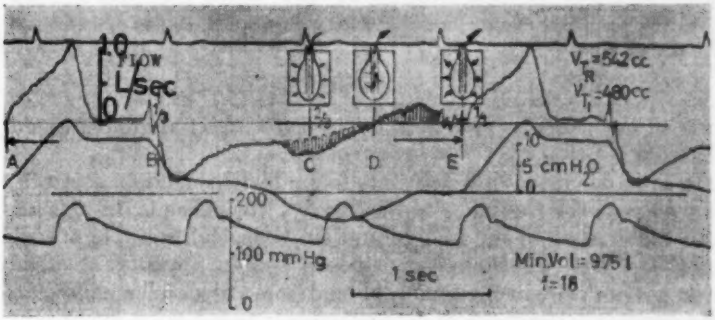


FIG. 6 Flow-pressure-characteristics for the Engoström Respirator obtained from a patient during anæsthesia. Note the variable pressure plateaux which differ from breath to breath according to changes in impedance. Observe also the phase of venturi as well as the phase of filling of the breathing bag. At the end of the 'filling' period the bag is under atmospheric conditions and allows the patient to take a spontaneous breath or to compensate passively for changes in FRC brought about by the venturi.

The high end-flow rate of gas relieves the patient of dyspnœic sensation, which is of importance in patients with serious cardiopulmonary disorders.

The amount of ventilation to be delivered to the patients is read from a practical ventilation nomogram constructed for the respirator[9]. The nomogram has been found to be correct with few deviations for

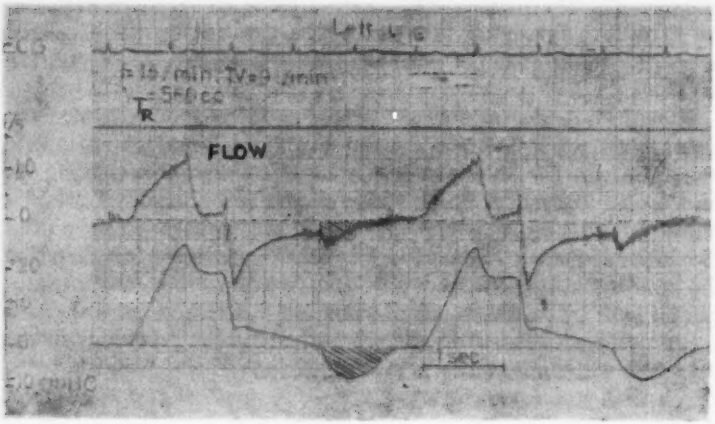


FIG. 7 Flow-pressure-curves from the left lung during ventilation with the Engström Respirator. Notice the acceleration of flow and the action of the venturi during expiration.

most of our patients (FIG. 8) as controlled with serial arterial blood analyses[1].

In some conditions it is necessary to use high insufflatory pressures to overcome the resistance of the airways, for example, in patients with constriction of the trachea and the bronchial tree. The water-safety-lock can be excluded in such cases and the pressures obtained in the patient's circuit can directly be read from a precision manometer. The expiratory resistance of the apparatus is very low – a few mm of water – and one may, therefore, wish to increase the resistance to expiration in patients with pulmonary œdema or fluid lungs. This is achieved with an expiratory valve resistance.

The ventilation received by the patient can be directly read from a spirometer in the expiratory circuit and is not influenced by the negative phase during expiration.

Humidification

Patients with tracheostomies tend to have dry airways. This is especially the case with children. During controlled ventilation with the Engström Respirator the patients receive a 100 per cent humidified gas mixture. Additional measures to ensure humidification are not necessary, except in patients who are brought in for treatment of tracheo-bronchitis, when in the beginning of the respirator treatment irrigation of the airways with saline or sodium bicarbonate solutions may be necessary.

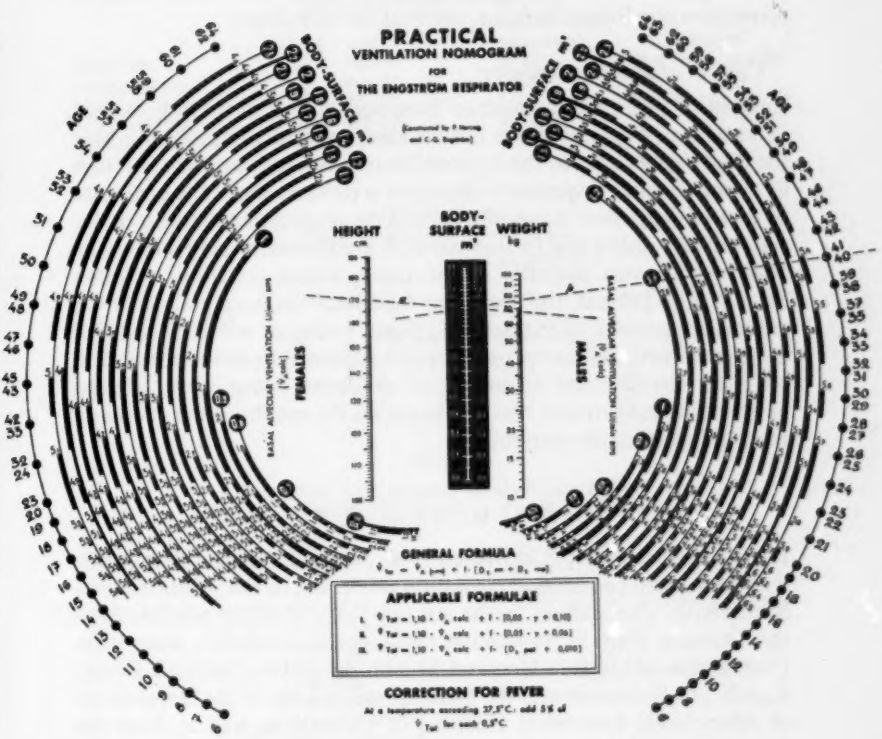


FIG. 8 The Ventilation Nomogram for the Engström Respirator.

Sedation

Conscious patients with respiratory distress and inadequate ventilation are very often restless and agitated. When the patient is connected to the respirator, it is, therefore, advisable to sedate him with an opiate or a barbiturate. The employment of opiates, usually contra-indicated in respiratory depression, is, when using controlled ventilation with the respirator, of positive value. Especially after accidental or surgical trauma to the chest, morphine in large doses has proved to be excellent. It relieves the patient of pain and facilitates synchronisation with the respirator. Synchronisation with the Engström respirator is easy and

it is very seldom that muscle relaxants have been necessary, except in convulsive conditions (tetanus, cerebral air-embolism).

Control of acid-base balance

Control of pH, pCO_2, standard bicarbonate and of arterial oxygen saturation or oxygen tension is advisable, especially in patients where the underlying cause to the hypoventilation is not clear and a mixture of metabolic and respiratory disorders is present. Once the acid-base picture of the patient is established and the respirator treatment under way, usually only a few further controls are necessary except in severe cases. In the vast majority of our cases, where metabolic acidosis has not been present, the respirator treatment has been guided by the clinical appearance of the patients. When young or newborn children are treated with the respirator, frequent adjustments of the ventilation are often necessary and the amount of ventilation administered is based on an empirical formula which is based on the weight of the child and the dead space of the respirator.

MATERIAL

During the period 1955 to March 1960, we have treated 522 patients with prolonged controlled ventilation according to the principles outlined above. The material in the last two years included patients from the Thoracic Units in Stockholm and Uppsala, pædiatric cases from Crownprincess Louisa's Hospital, Stockholm and the Pædiatric Clinic, Karolinska Sjukhuset and the Post-operative Unit of the Department of Anæsthesia, University Hospital of Uppsala as well as from the Renal Clinic, University of Lund.

The material is presented in TABLES 1 to 7.

Table 1

Respirator treatment

INDICATIONS	NO OF CASES	AGE YEARS	DURATION OF TREATMENT DAYS	RESULTS SURVIVALS	DEATHS
Lung and œsophagus	191	0.5–71	1–70	144	47
Heart	97	$\frac{8}{12}$–70	1–36	50	47
Non-chest	202	6–85	1–13	60	142
Infants and children	32	1 day–11 years	1–90	16	16
TOTALS	522			270	252

Table 2

Lung cases treated by respirator

DIAGNOSES	NO OF CASES	AGE YEARS	DURATION OF TREATMENT DAYS	RESULTS SURVIVALS	RESULTS DEATHS
Lung resections	163	0.5–71	1–70	123	40
Mult. rib fractures	14	26–70	1–17	10	4
Tracheo-bronchitis	6	0.5–3.5	2–9	6	0
Oesophageal resections	8	12–72	2–10	5	3
TOTALS	191			144	47

Table 3

Heart cases treated by respirator

DIAGNOSES	NO OF CASES	AGE YEARS	DURATION OF TREATMENT DAYS	RESULTS SURVIVALS	RESULTS DEATHS
After open heart op <3 years	13	2/12–27/12	1–4 hours	0	13
After open heart op > years	40	3–59	1–28	22	18
After closed heart op	44	3–70	1–36	28	16
TOTALS	97			50	47

Table 4

Operations for pulmonary tuberculosis – 1958
Patients requiring post-operative controlled ventilation

NUMBER OF PATIENTS	AGE MEAN	AGE RANGE	OPERATION	DURATION OF CONTROLLED VENTILATION (DAYS) MEAN	DURATION RANGE	RESULTS SURVIVALS	RESULTS DEATHS
8	51	38–64	Pneumonectomy	10	2–27	5	3
2	43½	42–45	Pneumonectomy + thoracoplasty	7	4–10	2	—
3	47	32–60	Lobectomy	5	2–8	3	—
1		13	Right upper lobectomy after left pneumonectomy	8		1	—
2	51½	39–64	Lobectomy + roof thoracoplasty	2	1–3	1	1
3	43	36–57	Lobectomy + segm. res. + roof thoracoplasty	15	6–9	3	—
1		38	Segmental res. + decortication + thoracoplasty		7	1	—
2	44	37–51	Decortication	9½	8–11	2	—
1		35	Decortication + thoracoplasty		8	1	—
1		30	Simultaneous bilateral upper lobectomy + decortication		10	1	—
1		31	Simultaneous bilateral decortication + left upper lobectomy		8	1	—
1		30	Simultaneous right upper lobectomy + resection of left upper lobe + segm. left lower lobe + left thoracoplasty		19	1	—
1		40	Simultaneous bilateral decortication + right-sided segm. resection + thoracoplasty		5	—	1
27						22	5

Table 5

Respirator treatment

DIAGNOSES	NO OF CASES	AGE YEARS	DURATION OF TREATMENT DAYS	RESULTS SURVIVALS	RESULTS DEATHS
Pulmonary œdema	7	14–70	1–7	6	1
MS + pulmonary hypertension	4	48–57	7	4	0
Cardiac arrest and massage	8	3–70	1–36	4	4

Table 6

Non-chest cases treated by respirator

DIAGNOSES	NO OF CASES	AGE YEARS	DURATION OF TREATMENT DAYS	RESULTS SURVIVALS	RESULTS DEATHS
Intoxications	13	22–63	1–14	7	6
Severe burns	3	22–53	1–7	1	2
Renal failure	142			43	99
Complication after general surgery	31	39–85	1–12	9	22
Cerebral trauma	13	6–77	1–13	0	13
TOTALS	202			60	142

Table 7

Intoxications treated by respirator

DIAGNOSES	NO OF CASES	AGE YEARS	DURATION OF TREATMENT DAYS	RESULTS SURVIVALS	RESULTS DEATHS
Tetanus	4	36–75	1–14	3	1
Barbiturate	6	22–58	1–7	3	3
Alcohol + œsophagus varicose bleeding	2	39–63	1–4	—	2
Snake poison (cobra)	1	43	2	1	—
TOTALS	13			7	6

RESULTS

The overall mortality with 252 deaths and 270 survivors can, at first, be looked upon as a poor result of our methods. It should, however, be underlined that this material includes many very ill patients and that some groups are very distorted. This is especially the case with the 'non-chest' group, which mainly consists of moribund patients with renal failure and with artificial kidney treatment.

CHEST SURGERY

The major part of the material is referable to chest surgery or inter-

ventions and trauma related to chest surgery. In this group we will find the most difficult cases from both circulatory and ventilatory points of view and in which the treatment has been most successful. In the combined chest-group, 288 patients have been treated with a survival of 194 patients (TABLES 2 and 3). Examples of the types of operations performed for advanced pulmonary tuberculosis are presented in TABLE 4. From this it is clear that many of the patients could not possibly have been operated upon without immediate respirator treatment. The indications for respirator treatment have been minimal lung-parenchyma in combination with mechanical interference with normal respiratory work. A large number of those patients also have had pneumonia, atelectasis and heart insufficiency (FIGS. 9A and B).

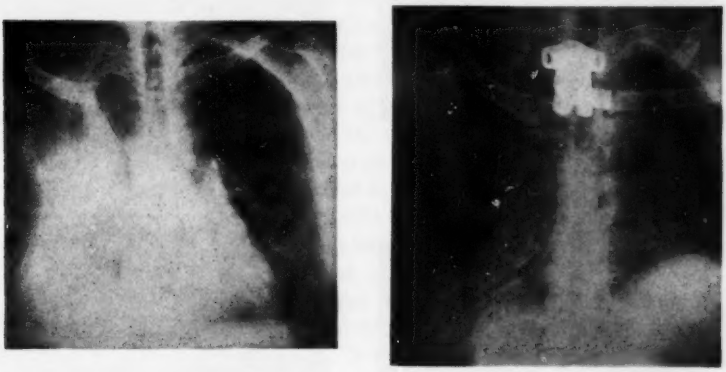


FIG. 9A and B Thirty-seven year old man. Right-sided pleura empyema and pleurocutaneous fistula with destroyed lung after blast accident ten years earlier. Pleuro-pneumonectomy + resection of the right chest wall was performed. Immediately after the operation the patient was tracheotomised and connected to the respirator. Severe paradoxical movements of the mediastinum and insufficient ventilatory exchange without the respirator. After two days stabilisation of the mediastinum and sufficient ventilation for short periods of time.

Trauma to the chest with mechanical instability of the thoracic cage and pulmonary contusion is a very important indication for primary tracheostomy and respirator treatment. This is nowadays a routine procedure at most Swedish hospitals[12]. Surgical fixation of fractured ribs is seldom carried out as it has been found that good stabilisation occurs in most patients after some days of respirator treatment. Larger dosages of opiates and sedatives are given to these patients to relieve them of the intensive pain. Injuries to the chest are often combined with serious cerebral trauma and some of the deaths occurring in our

patients have been due to that cause, or, as in one patient, a late acute rupture of an aortic aneurysm three weeks after the patient was taken out of the respirator.

An illustrative case is presented in FIGS. 10A, B, C and D.

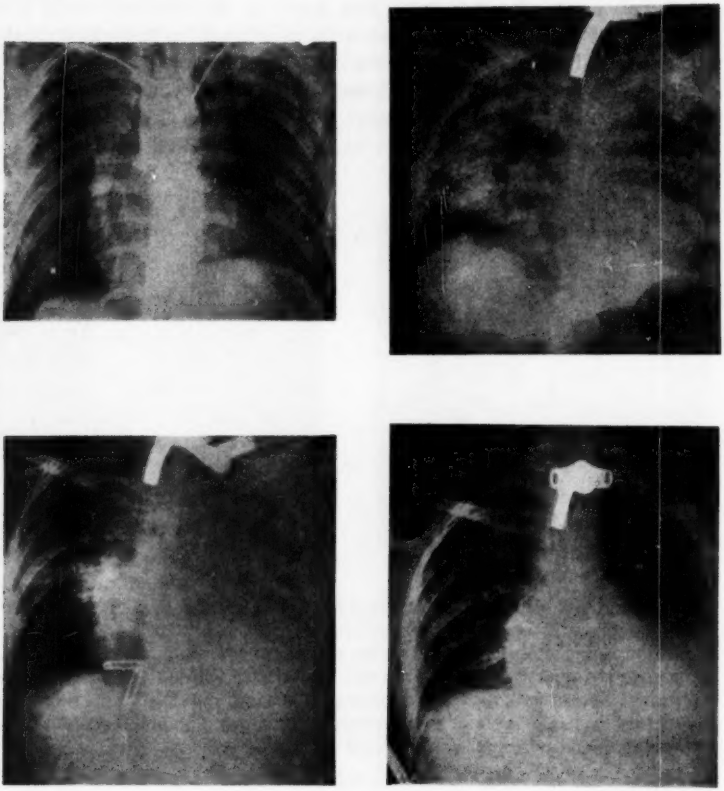


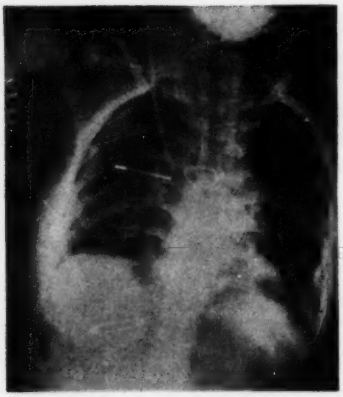
FIG. 10A, B, C and D Twenty-seven year old man run over by a car. On admission no signs of respiratory difficulties. Slight subcutaneous emphysema of the left chest. X-ray showed fractures of six ribs on the left side and fracture of the sternum. Pneumothorax and fluid on the left side. Infiltration of the pulmonary parenchyma. After twenty-four hours of clinically adequate ventilation acute ventilatory insufficiency with loss of consciousness. Tracheostomy and controlled ventilation for seven days with repeated suction of fluid from the left chest. Rapid improvement soon after the respirator treatment had been instituted. Discharged in good condition eight weeks after admission.

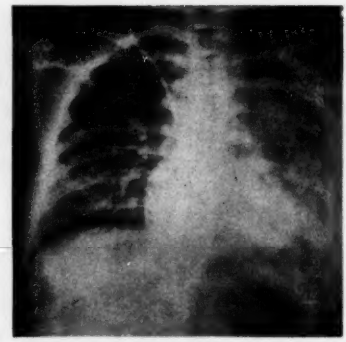
Chronic respiratory insufficiency with acute exacerbation in patients with advanced chronic emphysema or reduced pulmonary parenchyma is an important indication for respirator treatment. In this way the patients may be tided over a dangerous period of uncompensated respiratory acidosis due to an acute infection of the lungs. The treatment has also been tried on ten patients with cor pulmonale in acute heart failure with good results[2].

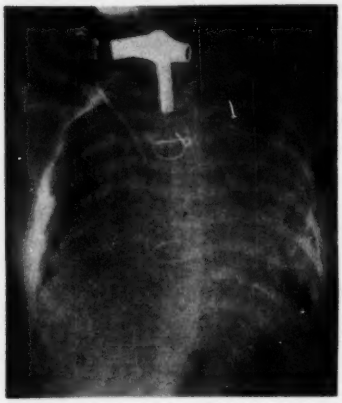
HEART SURGERY

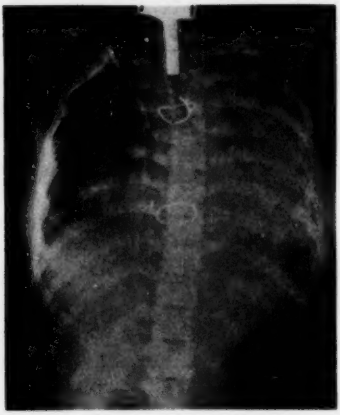
In 1954 Crafoord and Senning[18] performed the first successful operation with the aid of extra-corporeal circulation in Sweden. Since that time patients with severe post-operative pulmonary complications or failing circulation after closed or open heart surgery have constituted another large group of patients where controlled ventilation has been especially indicated. As can be seen from TABLES 3 and 4, many of these patients have succumbed during or immediately after the operation and could as well be labelled operative deaths. Such patients are omitted in most statistics, but we have included them in order to show the severity of the treated material. The possibility of controlled ventilation post-operatively makes it possible to keep many of these patients alive much longer than otherwise would be expected and in some cases some startling survivals have been observed. The indications for post-operative ventilation to these patients have been hypoventilation due to general fatigue or post-operative shock, failing circulation and in many patients pulmonary œdema, as in patients with pulmonary hypertension[27]. It thus can be noted that six out of seven patients with severe pulmonary œdema have survived. The condition of these patients was extremely bad and ordinary medical measures had failed to improve their status. Within thirty minutes to one hour the controlled ventilation had diminished or completely abolished their œdema.

In some patients after extra-corporeal circulation, prolonged controlled ventilation has been used post-operatively as a prophylactic measure. Such was the case in one patient with complete transposition of the great vessels in whom it was possible to carry out a full correction[20] and in one patient where parts of the coronary arteries were opened and replaced with a venous graft during a three hours perfusion[21]. Loth patients survived and are in good condition two years later. Controlled ventilation with good results has also been used after extracorporeal circulation in patients with Fallot's Tetralogy with earlier shunt operations[17 18] and with severe pulmonary changes post-operatively. FIGS. 11A, B, C, D and E.









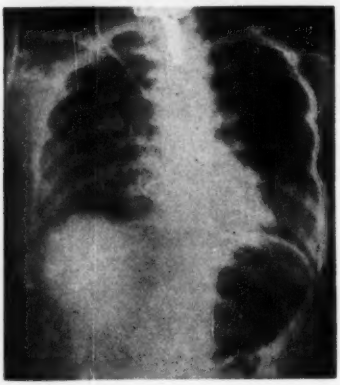


FIG. 11A, B, C, D and E Fifteen-year-old girl. Correction of Fallot's Tetralogy under extra-corporeal circulation. The first three post-operative days uneventful with normal blood-gas values. On the fourth post-operative day signs of arterial desaturation and increasing infiltrations of the lungs. Tracheostomy and respirator treatment for six days. Although 50 per cent oxygen in air was given by the respirator, arterial oxygen saturation was only 69 per cent and did not improve. It improved slightly on 100 per cent oxygen at the time of the most severe pulmonary infiltrations. Profuse secretions of œdematous type. From the fourth to sixth day gradual improvement. Discharged in good condition.

Recently it has been possible to extend our indications for mitral commisurotomy to include patients with pulmonary hypertension and gross pulmonary fibrosis. In these pre-operatively desaturated patients we perform tracheostomy at the operation and carry out respirator treatment until the patient can cope with the increased respiratory work. Even without the extra load due to thoracotomy, the respiratory work is very high in severe cases of mitral stenosis[16].

Severe tracheo-bronchitis, or mediastinal emphysema and tracheobronchitis as complications to acute tracheostomies in young children brought in to the hospitals for acute upper airway obstruction, are frequent and serious conditions with a high mortality[7]. Respirator treatment in combination with correction of the accompanying metabolic acidosis is the method of choice. These children are often exhausted by the respiratory effort and succumb in view of severe hypoventilation combined with dehydration and metabolic acidosis. Six children treated in this way have all survived and the treatment was in all cases instituted on vital indications on moribund patients. FIGS. 12A and B.

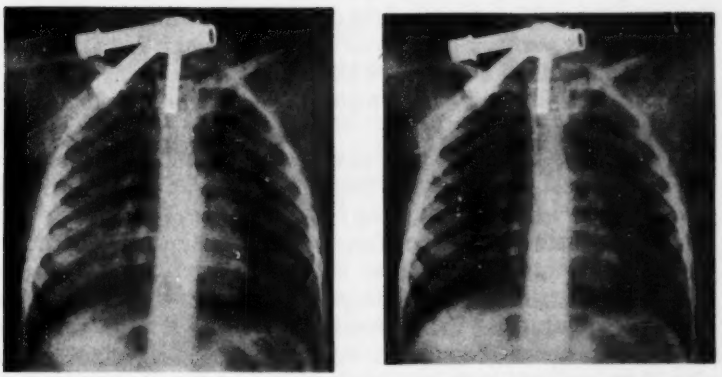


FIG. 12A and B A boy aged three years and four months with laryngeal œdema and inspiratory stridor. Tracheostomy. Twelve hours later respiratory difficulty with expiratory stridor. Restless and later unconscious with convulsions. Brought for respirator treatment. After irrigation and suction of the airways the patient was connected to the respirator. After three hours intensive treatment pH was 7.17, p CO_2 52mm/Hg and stand. bicarbonate 16.5 mEq. Before these analyses, the patient had received 1g bicarbonate i.v. After five hours respirator treatment and additional 1.5g bicarbonate i.v. the pH was 7.34, p CO_2 45mm/Hg and stand. bicarbonate 22.6 mEq. X-ray taken immediately after the patient had been put in the respirator showed a pronounced subcutaneous and mediastinal emphysema. After two days of respirator treatment most of the emphysema had disappeared.

RENAL FAILURE

Renal failure is frequently accompanied by pulmonary complications. In addition to electrolyte fluid disturbances the lungs are affected by bronchitis and stagnant secretions, bronchopneumonia, atelectasis, and fluid retention (fluid lung). Peritonitis and intestinal paralysis with distended abdomen also severely hamper the mechanics of respiration. Prolonged controlled ventilation in combination with dialysis has improved the results of treatment of renal failure. From 1956 to March 1960, 142 patients were treated in this manner at the Renal Clinic at the University of Lund. About 30 per cent of these patients could be saved in contrast to a 100 per cent mortality in the same types of patients according to clinical experience before the period of controlled ventilation[1024].

One of the most important indications for respirator treatment is the diminished arterial oxygen tension in those patients with fluid lung and the severe metabolic acidosis with its demand on increased excretion of carbon dioxide and thus an increased respiratory work. Oxygen therapy alone has not proved to be of value in these patients – it is necessary completely to take over the work of respiration. The results from the Renal Clinic have had the consequences that many hospitals now routinely put their patients on controlled ventilation as soon as renal failure is developing and by this treatment in combination with restricted fluid intake it has been possible to reverse the renal failure at an early stage.

It is thus evident that controlled ventilation should be instituted as soon as possible and not at that stage of development of the patient's disease when the chances to bring him back to health are nearly nil. This is illustrated from TABLE 6 (surgical complications), where almost all patients were moribund when brought over for respirator treatment. However, it was possible to prolong the lives of the patients by a few days. More and more doctors are now aware of the fact that the respirator treatment in seriously ill patients in need of acute surgery should be instituted early as a prophylactic measure. If started before the development of irreversible complications, the chances of survival will increase. This includes patients with infections (tetanus) or overdose of drugs (TABLE 7). As an example, two patients who died of barbiturate poisoning, were not brought for respirator treatment until cardiac arrest and heart massage had occurred.

Cerebral trauma constitutes a group with poor results, which naturally are due to the basic disease. By respirator treatment it is possible to keep patients alive for a few days more than otherwise, but the mortality is 100 per cent in this group. Patients with air-embolism as an accidental complication to open-heart surgery on the other hand

are most encouraging to treat with prolonged ventilation and two patients have been brought back to normal physical and mental states after more than three weeks respirator treatment.

Controlled ventilation of newborn and young children is relatively seldom tried and constitutes a field for further development. The reason for this is the difficulty in administering an adequate ventilation to such small patients over a long period of time and the problems of maintaining a free airway. With the Engström Respirator it is possible to give adequate volumes and the respiratory pattern of the respirator is also suitable for the newborn[13 24]. The number of patients treated in this group is not large and the results should be regarded as preliminary (TABLE 8). The indications have been very strict and all

Table 8

Infants and children treated by respirator

DIAGNOSES	NO OF CASES	AGE (WEIGHT)	DURATION OF TREATMENT DAYS	RESULTS SURVIVALS	RESULTS DEATHS
Compl. after tracheo-œsophageal fistula	8	1300–1600g	1–90	2	6
Cap. bronchitis pneumonia + empyema	7	1–25 days	1–5	1	6
Mediastinitis after œsophagoscopy	2	2.5–8 months	1–8	1	1
Anomalous thymus with phrenic nerv. paralysis	1	5 months	12	1	—
Ileus + peritonitis + metabolic acidosis	4	6–12 years	2–5	4	—
After closed heart op	7	1 day–11 years	2–112	4	3
Mult. rib fractures	2	5 years	5–16	2	—
Cong. gen. paralysis	1	1 day	90	1	—
TOTALS	32			16	16

children have had absolute need for respiratory assistance. As can be expected, the best results are obtained in those groups where somewhat older children are present, but that this kind of treatment can be successful is illustrated from two of eight newborn infants with severe complications after operations for tracheo-œsophageal fistulas. In one baby with general paralysis after birth, the respirator treatment has been going on for more than ninety days, with the child growing normally.

Four children with severe peritonitis and ileus combined with metabolic acidosis all survived after surgery and respirator treatment. This is also in agreement with our experience from patients with renal failure and ileus and post-operative ileus after abdominal surgery in adults. If the controlled ventilation is instituted at an early stage of the process, the chances for the patient are good.

CONCLUSION

The experience and the results from controlled prolonged ventilation with the Engström Respirator connected to a tracheostomy in patients with respiratory distress or impaired circulatory function have shown that this kind of treatment is life-saving for many patients. In a large number of conditions – post-operatively as well as pre-operatively – an increased amount of ventilatory effort is present. Many patients are not able to produce this amount of extra effort and, therefore, go into a hypoventilatory state with serious effects on circulation and metabolism. Such complications are apt to occur in thoracic surgery and in that field the use of controlled ventilation has had its widest application. Many operations for tuberculosis and tumors of the lungs as well as for acquired and congenital heart disease, could not have been performed without respirator treatment.

The respirator treatment has found new application in the treatment of severe pulmonary œdema which could not be reversed by ordinary measures.

In patients with renal failure undergoing dialysis treatment, controlled ventilation has improved the survival rate by 30 per cent. By early institution of controlled ventilation in patients with signs of failing renal function the number of dialyses has been shown to decrease.

Controlled ventilation of the newborn with surgical and neonatal complications has been most encouraging and has opened up a new field for treatment.

When applying controlled ventilation to a vast number of patients with serious disorders of the ventilatory and circulatory systems, a respirator of highest quality has to be used. The respirator should be able to deliver predetermined tidal volumes of high accuracy. The flow-pressure characteristics should be as advantageous as possible. This involves acceleration of the flow from zero-velocity to a high end peak velocity followed by a variable pressure plateau for optimal distribution and thus diffusion of gas within the lung parenchyma. Spontaneous ventilation should be possible without changes in the pre-set minute ventilation or the applied respiratory frequency. The machine must be completely dependable and possess monitoring features so that it can be used and attended by nurses and technicians without expert knowledge. According to our experience, the Engström Respirator fulfills these requirements and, therefore, is a most valuable tool in the treatment of severely ill patients.

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Experiences in a small respiratory unit of a general hospital

With special reference to the treatment of tetanus

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Until a few years ago the treatment of inadequate ventilation was largely in the hands of infectious fever specialists dealing particularly with spino-bulbar poliomyelitis. The poliomyelitis epidemic in Denmark in 1952[1,2] altered the situation immediately, for under these circumstances tracheostomy with intermittent positive-pressure respiration established its niche in present-day therapy. Since then anæsthetists have become aware of the number of other cases who could be saved by the simple expedient of avoiding anoxia and providing adequate artificial ventilation. Thus, central neurological respiratory depression due to injury, poisoning or other disease[3,4], polyneuritis[5], myasthenia gravis[6], tetanus[7,8,9], acute chest injuries[10] and respiratory insufficiency due to pulmonary infection or disease, especially in cases of chronic bronchitis and emphysema[11,12], have also been treated in this way.

Our experience in the past few years has been one of frustration in trying to deal with these problems in large hospital wards. There have often been difficulties in mobilising rapidly even the simplest resuscitative equipment, while the long-term treatment has involved the considerable problems of nursing and overall supervision of mechanical apparatus. Results were naturally unsatisfactory. It was because of these problems that it was decided to equip a small two-bedded unit for the treatment of respiratory insufficiency – from whatever cause. This project was sponsored by the Wessex Regional Hospital Board both for the treatment of patients in the Southampton district and from surrounding hospital areas. Patients were then transferred from other wards or from their homes. In the latter cases a trained anæs-

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thetist, with all necessary resuscitative apparatus, went by ambulance to supervise the patient's journey to the Unit.

Such was the demand on this small bed space that it was still occasionally necessary to treat cases in the wards, so that after the first eighteen months the Unit was enlarged to a total of five beds.

The advantages of the Unit itself were immediately obvious and our experiences agree closely with the findings of other workers[13]. In the first place there was concentration of equipment, including suction machines, mechanical ventilators, intubation and bronchoscopic apparatus, which could never be achieved elsewhere and which could be kept in a constant state of preparedness. Furthermore, it was possible to maintain specialised nursing care at all times without hindrance to the adjacent ward work and disturbance of other patients.

The teamwork required must be of the highest order; skilled nursing attention and physiotherapy is foremost while overall supervision is given by a physician with an anæsthetist to manage the respiratory aspects of the case. Other members of the medical staff are called in consultation as required.

The cases treated since the Unit was opened in August 1958 are presented in TABLE 1.

Table 1

DISEASE	NO. OF CASES	TRACHEOSTOMY	IPPR	CURARISATION
Tetanus	14	11	9	9
Neurological	23	13	5	1
Respiratory	6	4	2	1
Post-operative	7	6	3	1
Poisoning	8	3	3	—
Miscellaneous	4	1	—	—
TOTALS	62	38	22	12

The neurological cases consisted of several disease entities and these are shown in TABLE 2.

Table 2

DISEASE	CASES TREATED	SURVIVED
Head injuries	6	3
Cerebral hæmorrhage	1	0
Subarachnoid hæmorrhage	1	1
Bulbar palsy	1	1
Brain stem lesion	1	0
Encephalitis	3	2
Pneumococcal meningitis	1	0
Status epilepticus	1	1
Polyneuritis	5	5
Myotonia	2	2
Residual poliomyelitis	1	1
TOTALS	23	16

Head injury cases were either returned from a neurosurgical unit following craniotomy, or otherwise required no surgical intervention. It will also be seen that cases of poliomyelitis, apart from one case of residual poliomyelitis, do not figure in this list; this is because these cases are normally treated at the Regional Poliomyelitis Centre at Portsmouth.

The respiratory cases included cases of cor pulmonale, chronic bronchitis, emphysema and asthma as well as staphylococcal bronchopneumonia. Respiratory insufficiency due to acute chest injuries such as 'stove-in-chest', or following pulmonary resections or cardiac operations, are not included as these are treated at the neighbouring Regional Thoracic and Cardiac Centre at Southampton Chest Hospital.

Of the post-operative cases of respiratory insufficiency, all were seriously ill, often moribund patients who had undergone emergency surgery. The clinical picture was usually complicated and the treatment of respiratory insufficiency was only part of a more generalised resuscitation.

Six of the eight cases of poisoning treated in this Unit were due to barbiturate overdosage and all survived. One case of insecticide poisoning required little in the way of active therapy and the only death in this group was in a child of two years following accidental ingestion of an overdose of primidone.

The miscellaneous cases consisted of a severe reaction to antitetanus serum, a case of arthritis of the temporo-mandibular joints – initially diagnosed as tetanus, a case of lupus erythematosis with cerebral manifestations including convulsions and œdema of the face, tongue and neck who died of renal failure, and a fourth case who died suddenly with an epileptiform type of convulsion for which no cause was found at post-mortem.

From TABLE 1 it will be seen that a very high proportion of these cases required tracheostomy with, or without artificial ventilation. This increases the work and supervision enormously in terms of working hours and is out of all proportion to the actual number of cases treated. TABLE 3 shows, however, that forty-one out of the sixty-two cases survived, most of the survivors making a complete recovery.

Table 3

DISEASE	NO OF CASES	SURVIVORS
Tetanus	14	10
Neurological	23	16
Respiratory	6	3
Post-operative	7	3
Poisoning	8	7
Miscellaneous	4	2
TOTALS	62	41

Of these cases probably the most complex and difficult to handle are those of severe tetanus. Of the fourteen cases admitted to the Unit, nine were severe and were having frequent generalised spasms (type 3 in Knott and Coles classification)[14] and five were mild, exhibiting rigidity and trismus (type 2 in Knott and Coles classification), although one of the five also had laryngeal changes. Four of the five mild cases made an uneventful recovery. Two of the mild cases required tracheostomies for severe concurrent chest infections and one also had laryngeal stridor. The latter case, a severely disabled emphysematous patient with a purulent bronchitis and severe asthma, died suddenly in cor pulmonale.

Of the nine severe cases, three succumbed; the first was due to inexperience on our part, the tracheostomy being made too low and the patient dying on the fifth day of hæmorrhage from the innominate artery. The second was an old man of seventy years, who presented with the quadruple pathology of tetanus, cerebral arteriosclerosis, chronic bronchitis and emphysema, and chronic alcoholism. He was treated initially with sedation and tracheostomy, but later required curarisation and artificial ventilation to control his spasms. He died on the eighth day of treatment from an overwhelming chest infection. The third case was a woman of fifty-seven years who succumbed to a large pulmonary embolus after one week of IPPR treatment.

These cases provide every form of problem imaginable. Sedation plays a great part and the value of chlorpromazine and the barbiturates is well recognised[15]. Nevertheless, it is our experience that in the really severe cases sedation is not sufficient. Spasms are still initiated on the slightest stimulus making nursing attention, physiotherapy and feeding difficult or even impossible, while with the deepest sedation there may be disorders of ventilation and concurrent pulmonary stagnation and infection. In the majority of cases of tetanus admitted to the Unit treatment has commenced with sedation, using chlorpromazine either alone or with a barbiturate, but each severe case has given rise to such anxiety that nowadays we breathe a sigh of relief when the decision is made to perform tracheostomy, curarise the patient and carry out artificial ventilation. In this way it is quickly possible to get the situation firmly under control again – laryngeal spasm is eliminated, generalised spasms and temperature are controlled, ventilation is regulated and frequent changes of posture, chest physiotherapy and tracheobronchial aspiration can be carried out. Feeding difficulties are also eliminated by the use of a plastic nasal intragastric tube. This régime is not undertaken lightly as it may go on for a period of two to three weeks or more and increases the work and supervision required to an enormous extent. Clearly these techniques are impracticable in many centres, often, unfortunately, in those areas where tetanus is most pre-

valent. In our opinion, however, where the facilities and trained staff are available this method offers the severest cases of tetanus a very high chance of recovery.

Some of the special problems in these cases can be discussed under separate headings:

THE CONTROL AND MANAGEMENT OF PULMONARY VENTILATION

To control the spasms and to allow artificial ventilation to be carried out d-tubocurarine chloride is used as the relaxant of choice and is given intramuscularly – usually hourly – either with, or without the addition of hyaluronidase. Other workers[7,8] have used suxamethonium drips, but this has all the disadvantages of prolonged intravenous infusion therapy.

Several ventilators have been used, chiefly the Radcliffe 'Positive-Pressure' Respiration Pump with cam-operated valves[16], the Radcliffe 'Positive-Negative' Respiration Pump[17], the Beaver Mark II[18] and also the Blease Pulmoflator, Model P.3[19] with negative phase and patient-triggering mechanisms. A humidifier is used in conjunction with all of these machines. The minute volume requirements of the patient are estimated on a weight basis, using Radford's Nomogram[20] and this is measured on the expiratory side of the pump by means of a Dräger Volumeter, or Wright Anemometer[21]. In tetanus, even with small amounts of sedation, once IPPR is commenced the patient often passes into a sleep-like state and consequently ventilation has to be monitored on clinical and biochemical observations. Changes in skin texture and colour, pulse rate and blood pressure are particularly important and are recorded on a twenty-four hour chart together with serial recordings of the minute volume and inflation pressure readings. In conjunction with this a few serial estimations of arterial pH, oxygen saturation and pCO_2 are carried out. The pCO_2 is measured in two ways: (*a*) using Astrup's technique[22], or otherwise (*b*) indirectly, by measuring the pH and blood CO_2 content and calculating pCO_2 from the Henderson-Hasselbalch equation.

These methods appear to be acceptable for clinical purposes providing that the sources of error are recognised[23]. If it is considered inadvisable to perform repeated arterial punctures then 'arterialized' venous samples are employed[24]. It may take some hours to settle a case of tetanus on IPPR and ventilation is normally monitored for the first few days, but after this it is only necessary to determine blood gas tensions intermittently. In general, however, some over-ventilation is employed and an attempt is made to achieve a pCO_2 of 25–30mm/Hg.

To prevent pulmonary complications frequent changes of posture are required. This is combined with chest percussion, expiratory vibra-

tion and careful tracheo-bronchial aspiration via the tracheotomy tube. The latter manœuvre must be performed under aseptic conditions using a sterile catheter on each occasion and introducing the catheter by means of sterile forceps. With this treatment, adequate humidification and appropriate chemotherapy as indicated, lobar collapse or consolidation can be reduced to a minimum. Frequent serial chest X-rays are not necessary and in only two of these long-term cases has bronchoscopy been necessary.

During the recovery stage when generalised spasms have disappeared, usually after two to four weeks of therapy, curare is omitted and reversal of curarisation is carried out. Edrophonium is given initially and when it is quite clear that generalised spasms are no longer occurring the reversal is completed by injections of atropine followed by neostigmine. At this stage, with the return of spontaneous respiration, the patient awakens immediately and it is often necessary to give further sedation for this may be the first recollection he has of such an illness; this is obviously particularly important in the case of children.

Despite reversal of curarisation with anticholinesterase drugs spontaneous ventilation is often inadequate for many hours and it may be necessary to provide assisted ventilation with a patient-triggered ventilator for perhaps twenty-four hours.

TRACHEOSTOMY: ITS MANAGEMENT AND COMPLICATIONS

The indications for tracheostomy in tetanus have been well outlined by Ablett[25]. The main indications for its use in tetanus in our Unit have been:

1 As a prophylactic measure in severe cases.

2 To obviate the dangerous effects of laryngeal spasm which may accompany the generalised spasms.

3 To prevent the inhalation of mucus or regurgitated stomach contents.

4 To permit tracheo-bronchial toilet, thus preventing pulmonary collapse and infection.

5 To enable prolonged IPPR to be carried out with the use of muscle relaxants.

We would agree wholeheartedly with other workers that early tracheostomy is advisable in tetanus[26 27].

Tracheostomy is always performed under general anæsthesia with endotracheal intubation. In this way the surgeon is able to make an unhurried dissection and remove a diamond or square-shaped portion of the trachea at the level of the second and third rings.

Rubber, cuffed tracheotomy tubes are used initially, either of the

Oxford pattern, Morrant-Baker or James' types. If a rubber, cuffed tracheotomy tube is required without IPPR humidification is provided by means of a fan-blower made by our own hospital engineers and a thermostatically controlled water bath. The cuff of the tube is deflated at four-hourly intervals, for a period of five to ten minutes in the head-down position, to prevent ulceration of the tracheal mucous membrane. Each tube is changed at weekly intervals.

In the convalescent stages of the disease and when normal oral feeding has been re-established the rubber, cuffed tube is replaced by a silver Durham's tube with a slit in the upper curve. This is corked, intermittently at first, so that the upper airway and laryngeal function are re-established. When this is satisfactory the tube is removed and a firm dressing is applied to the tracheostome which closes down rapidly and usually heals within a week.

COMPLICATIONS

1 *Obstruction of the tube* is usually due to inadequate humidification and the accumulation of inspissated secretions.

2 *Accidental displacement of the tube.* Great care is needed for the first twenty-four to forty-eight hours after tracheostomy until a track has formed as displacement of the tube at this stage is a serious matter. It is, therefore, a wise precaution to see that the tapes attached to the tube are firmly secured round the patient's neck and that there is no tension from other tubing.

3 *Faulty tracheotomy tubes.* None of the tubes in current use is absolutely ideal and occasionally faults in manufacture occur. The cuffs may burst or leak slowly, or faulty vulcanisation at the neck of the tube give rise to serious leaks which may have to be remedied immediately.

4 *Infection.* It is difficult, in our experience, to prevent secondary infection both at the tracheostome and of the tracheobronchial tree, however meticulous the aseptic aspirating technique may be. This infection has to be covered by appropriate chemotherapy.

5 *Tracheal granuloma.* This has been seen on two occasions and was severe enough to cause tracheal obstruction when the tracheotomy tube was removed[28]. In both cases the tracheostomes were closed successfully after removal of the granuloma.

6 *Sinuses and scars.* Most sinuses eventually close, but as a result of pressure and infection at the tracheostome some patients are left with keloidal scars. Although these may appear unsightly at first, many of them disappear in the course of time and the number which require plastic surgery at a later date is extremely small. In any case we feel this is a small price to pay for the successful outcome of their treatment.

CONTROL OF FLUID BALANCE AND NUTRITION

Basically, an adequate daily intake of 2,500 calories and 2.5–3 l of fluid must be achieved for an adult assuming that renal function is adequate.

In mild cases of tetanus there may be trismus and difficulty in swallowing in which case adequate nutrition can only be achieved by the use of a plastic nasal intragastric tube. In the most severe cases requiring tracheostomy and IPPR a rubber, cuffed tracheotomy tube seals off the tracheo-bronchial tree against accidental regurgitation and inhalation of gastric contents. These cases often present with a concomitant paralytic ileus and intravenous infusion may be necessary for twenty-four to forty-eight hours, but as soon as bowel activity recommences – as determined by auscultation of the abdomen and assessment of gastric residue – a gradual change over is made to intragastric feeding via a plastic œsophageal tube. In this way the difficulties of long term intravenous infusion therapy are avoided. Intragastric feeding, using a balanced foodstuff such as Complan, with an adequate fluid intake ensures a normal electrolyte balance even after weeks of therapy.

In tetanus, providing pulmonary ventilation is correctly maintained, the only abnormal electrolyte changes found have been occasional alterations in the serum potassium level and alterations in the blood urea concentration. In the early stages of the disease, perhaps with intense muscle activity, or with some initial hyperventilation if IPPR is performed, it is not uncommon to find a lowered serum potassium level; this is of significance if artificial ventilation is being carried out since this alters the amount of relaxant required. A raised blood urea level of 40–80mg per cent seems universal in these cases and is probably a reflection of impaired renal function or excess urea formation[29].

CONCLUSIONS

Some of the problems which occur during the management of patients suffering from respiratory insufficiency have been discussed and particular reference has been made to the treatment of tetanus.

The successful outcome of treatment depends entirely on the teamwork and facilities available and a special plea is made for the introduction of respiratory units in all busy general hospitals.

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A new mechanical ventilator

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Since the discovery of the relaxant drugs, many techniques requiring controlled ventilation have been introduced. The method most commonly used is the rhythmical squeezing of a bag by the anæsthetist. This paper describes the development of an automatic 'bag squeezer' which fulfils the following requirements. (1) The ventilation should be designed to minimise the cardiovascular effects of intermittent positive pressure on the anæsthetised patient. (2) The machine should provide the anæsthetist with as much information as possible about the ventilation and the degree of relaxation of the patient. This will then largely compensate for the loss of contact with the bag. (3) The machine itself should be sufficiently small to stand on an anæsthetic apparatus. (4) It should operate from the gases supplied without ancillary sources of energy.

When a Boyle's or similar anæsthetic apparatus is used with a non-rebreathing circuit, the flow of gases from the apparatus will be equal to the minute volume delivered to the patient provided no loss of gas occurs.

The gases leave the reducing valves on a modern continuous flow apparatus at a pressure in excess of 5lb/square in (352cm water) and pass into the flowmeter through a needle valve. From this, gas at a pressure below 30cm water is delivered to the patient. Hence a pressure difference of over 300cm water is available as a source of energy. The mechanism and valves of the automatic ventilator are operated entirely by a part of this pressure difference. The valves are of a type that needs no lubrication and they are not affected by condensation of water vapour from the expired gas. The expiratory valve, together with the whole of the expiratory side of the apparatus, has been made readily detachable for cleaning and sterilisation.

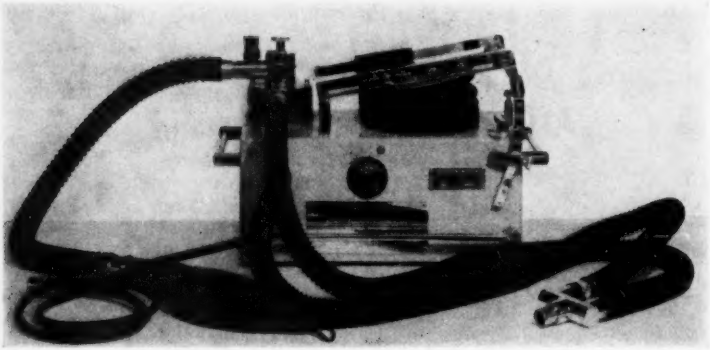

FIG. 1 The ventilator

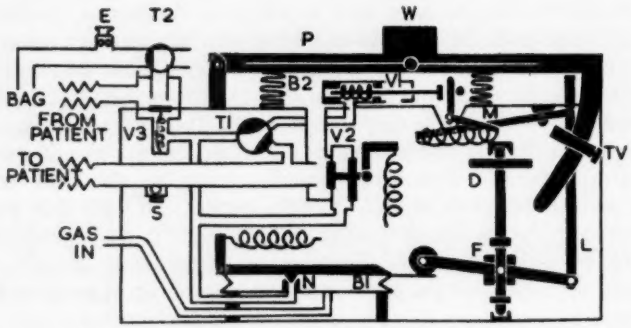

FIG. 2 Diagram of the ventilator showing mechanism

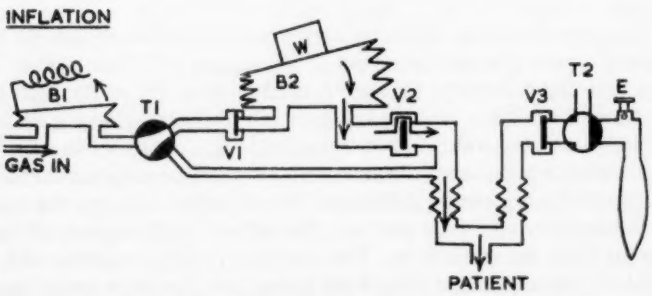

FIG. 3 Diagram of the ventilator showing gas flow during the inspiratory phase

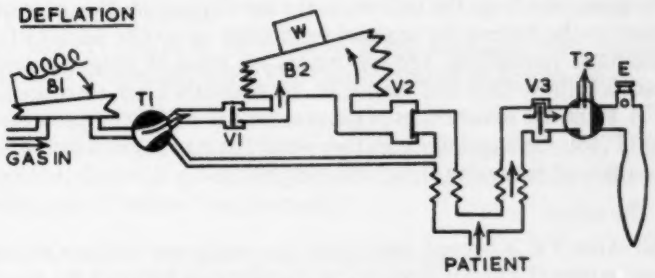

FIG. 4 Diagram of the ventilator showing gas flow during the expiratory phase

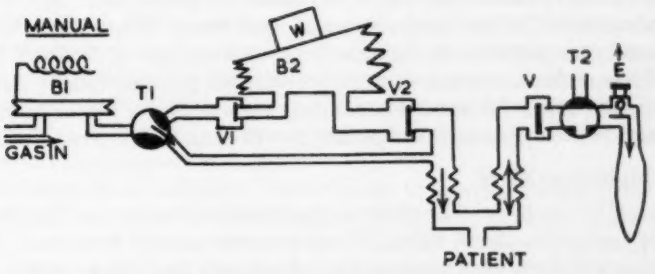

FIG. 5 Diagram of the ventilator showing gas flow with manual ventilation

The ventilator produces inflation with a variable weight-loaded bellows (B2) delivering a pre-set tidal volume through inspiratory and expiratory disc valves (V2, V3) in a non-rebreathing circuit. The weight (W) is movable along a graduated scale (P) to give pressures of 8–30cm water. In addition, it has a second stage consisting of a bellows and valve (B1 and V1) to provide 'time' cycling for the end of the inspiratory phase and to provide the operating pressure for the inspiratory and expiratory disc valves. It also has a tap (T1) to divert the gas flow direct to the patient's lungs for manual ventilation with a bag. The gas flow from the anæsthetic apparatus is admitted to the bellows (B1) and kept at a constant pressure of 100cm water by spring loading. The pressure in the bellows is maintained when it is empty by the valve (N) that regulates the outflow when the two end plates of the bellows converge on each other. A constant back pressure on the rotameters is thereby provided to prevent the bobbins from jumping up and down.

The gases pass from the bellows to the tap (T1) which diverts the flow direct to the patient for manual ventilation or to the valve (V1) for automatic ventilation. The gas passes via valve (V1) to the weight-loaded bellows (B2) and thence to the patient's lungs through valve (V2). From the patient's lungs gas reaches valve (V3) and proceeds via tap (T2) to the bag and expiratory valve for manual ventilation or to the atmosphere during automatic ventilation.

The valves

The valve (V1) is opened and closed by a toggle mechanism (M) and a lever system (L) that is acted on by the alternate filling of the two bellows. Valves (V2 and V3) are operated automatically by the changes in pressure in the tube from bellows (B1) to valve (V1) which occur when valve (V1) is opened and closed. This is achieved by spring biased diaphragms in small pressure chambers attached to the valves (V2 and V3). These are set so that valve (V2) opens at a pressure of 75cm water and valve (V3) closes at a pressure of 50cm water. When valve (V1) is closed, the pressure in the chambers rises to that in bellows (B1) (100cm water) and when valve (V1) opens, the pressure falls to that in bellows (B2) (8–30cm water). A safety blow-off valve (S) set at 35cm water, is incorporated in the patient circuit as an added protection.

Inspiratory phase

Valve (V1) is closed. The fresh gas is retained in the bellows (B1) which fills during this phase. Valve (V2) is open and valve (V3) is closed. The weight (W) forces the gas from the bellows into the patient's lungs and produces inflation. The inflation pressure depends on the position of the weight (W) on the graduated scale (P). The tidal volume depends on the setting of the stop on the tidal volume scale (TV) and the duration of this phase depends on the height reached by the bellows (B1) before it causes the lever system (L) to open valve (V1). This is set on the 'duration of inflation' control which alters the height of the fulcrum (F).

End of inspiratory phase

When valve (V1) opens, the pressure in the chamber falls and valve (V2) closes and valve (V3) opens.

Expiratory phase

The patient's lungs are now open to the atmosphere and expiration takes place. The gas in the bellows (B1) together with the flow of fresh gases and the small volumes from the pressure chambers now pass through valve (V1) into bellows (B2) and fills it. The duration of this phase depends on the height reached by the bellows (B2) before the lever system closes valve (V1).

End of expiratory phase

When valve (V1) closes, the pressure in the chambers rises, closing valve (V3) and opening valve (V2), enabling inflation to take place.

Manual ventilation

Taps (T1) and (T2) are turned to the position for manual ventilation. The patient circuit is now a Mapleson D semi-closed system (FIG. 5) allowing partial rebreathing to occur.

THE VENTILATOR IN USE

The minute volume required is set on the rotameters of the anæsthetic apparatus and the desired tidal volume is set on the tidal volume scale. The frequency of ventilation will be equal to the minute volume divided by the tidal volume. The weight is adjusted to provide just enough pressure to enable the tidal volume to be inflated, which depends on the compliance of the patient's lung and chest wall. Compliance is partly dependent on relaxation and any alteration will show as a change in the volume inflated as the pressure remains constant. Such a volume change is immediately evident to the anæsthetist who may wish to compensate the change by varying either the inflation pressure or the degree of relaxation of the patient.

The 'duration of inflation' control is set to provide sufficient time for inflation to occur, which depends on the airway resistance. Increase of resistance during anæsthesia will show as a failure of the tidal volume to be inflated in the time set. Clearly the machine allows the anæsthetist to distinguish changes in the airway resistance arising due to obstruction of the airway or accumulation of secretions, from changes of compliance associated with the degree of relaxation.

At all times information is immediately available concerning the minute and tidal volumes, the inflation pressure, the compliance and airway resistance during anæsthesia. The anæsthetist may at any moment change to manual ventilation with a bag to 'feel' conditions in the patient.

A mounting for a Wright Anemometer is provided on the expiratory valve to measure the volume expired if this is needed.

Ventilator performance

One object of the present machine was to produce adequate positive pressure ventilation with the least physiological disturbance of the patient. It is desirable to provide a high flow to the patient early in the inspiratory phase to enable inflation to take place in a relatively short time. The result of this characteristic is twofold. Firstly, pulmonary blood flow suffers least interference as the intra-alveolar pressure is then atmospheric for a relatively large proportion of the respiratory

cycle. Secondly, the venous return to the right side of the heart is not impeded.

The performance of the ventilator was investigated by measuring tracheal flow, tidal volume and mouth pressure in anæsthetised patients.

Flow rates were measured by using a Fleisch pneumotachograph with a Statham PM 197TC differential pressure transducer of range ±0.7cm water connected to a carrier amplifier (Southern Instruments MR 501). Tidal volume was derived from the flow signal by electrical integration. Mouth pressures were measured by a Statham P23G transducer and carrier amplifier (as above). The pen recorder was Siemens Ediswan Pen Oscillograph MK III.

RESULTS

FIG. 6 shows the results from a female patient aged forty-eight years undergoing a cholecystectomy. The patient was apnœic, the abdomen was open and relaxation was complete while the recordings were made.

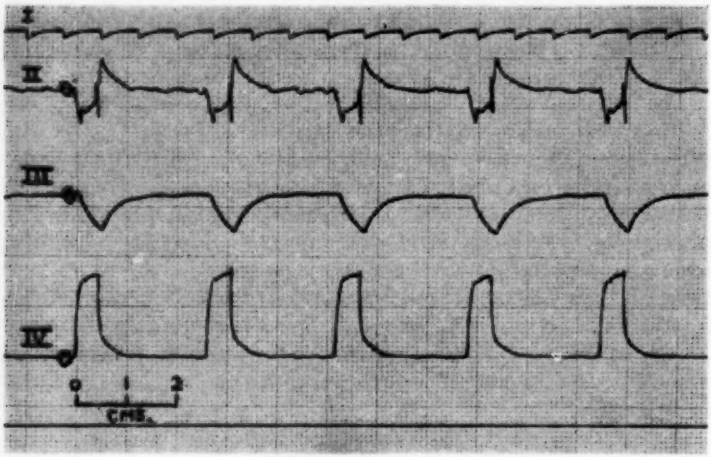

FIG.
Trace I is a time tracing in seconds
Trace II is the tracheal flow. Scale 1cm = 200 l/min flow
Trace III is the tidal volume. Scale 1cm = 1 litre
Trace IV is the mouth pressure. Scale 1cm = 10cm water

Consider tracing II. Flow into the patient corresponds with a downward deflection of the pen and a tracheal flow rate of over 120l/min is reached at the commencement of the inspiratory phase. The flow rise

time is 0.2secs. This rapid rise in flow rate results from the low resistance of the inspiratory valve. The high flow delivered by the machine causes a rapid rise in the mouth pressure (IV) which produces a tidal volume (III) of 700ml in 0.7 sec. *i.e.* approximately 20 per cent of the respiratory cycle. It may be noted that the extra dead space introduced by the pneumotachograph was 200ml and that under normal operation the tidal volume inflated would have been 500ml and this would have been inflated in 0.4secs or approximately 12 per cent of the respiratory cycle.

Trace II also shows that a flow rate in excess of 120 l/min was achieved at the commencement of expiration. With these high flow rates it is seen that the alveolar pressure is held at atmospheric for a duration of two seconds in a respiratory cycle of three and a half seconds as shown on traces II and IV when mouth pressure is atmospheric and there is no tracheal flow. This form of ventilation causes little physiological disturbance and performs controlled ventilation in a way not possible with a hand squeezing a bag.

The prototype shown in FIG. 1 has been in routine use for some months in the Department of Anæsthetics at Westminster Hospital. Over one hundred cases ranging in age from eight to eighty-six years have been ventilated with this machine and no disadvantages have been found. A production model is undergoing final clinical trials.

VENTILATOR SPECIFICATION

Minute volume	2–20 l/min
Tidal volume	200–800ml
Frequency of ventilations	10–60 per min
Inflation pressure	8–30cm water
Expiratory/inspiratory time ratio	2–1 to 10–1
Resistance of expiratory valve and tap	less than 1cm water for 60 l/min flow
Peak inspiratory flow rate (Wright peak flow meter)	110 l/min at 10cm water 140 l/min at 20cm water 180 l/min at 30cm water

SUMMARY

A new mechanical ventilator is described for intermittent positive pressure ventilation during anæsthesia. It is operated by the gas flow from a Boyle's or similar anæsthetic apparatus and requires no ancillary source of energy. Results are presented to show how the requirements for this type of ventilator may be realised in clinical practice.

Acknowledgements

I wish to thank Dr G. S. W. Organe, Director of the Department of Anæsthetics for allowing the ventilator to be used by members of his department at Westminster Hospital and Dr P. Cliffe, Director of the Department of Clinical Measurement, for his help in the investigation of the ventilator's performance.

I also wish to thank Mr J. Blease (Blease Anæsthetic Equipment Ltd) for his continued help in developing the production model of the ventilator.

Circulatory and respiratory effects of ether, halothane and the azeotrope mixture *

A comparative study in children

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The cardiovascular and respiratory depressant properties of anæsthetic concentrations of halothane have been demonstrated by Raventòs[1], Johnstone[2], Hall and Norris[3] and others. An azeotropic mixture of halothane and ether has been described by Hudon, Jacques and Boivin[4] who claim that the addition of ether modifies these depressant effects. This was not the experience of Raventòs and Dee[5] who found that the azeotrope caused comparable degrees of bradycardia, hypotension and respiratory depression to halothane in the dog. However, Dobkin and his colleagues were impressed by the absence of depression found with the mixture in man[6] and in the dog[7], as compared with halothane. It may well be that species variation accounts for these conflicting reports and so it was thought desirable to investigate the problem further in the human subject.

Some physical characteristics of halothane and the azeotrope are shown in TABLE 1.

Table 1

Physical characteristics

	HALOTHANE	AZEOTROPE
Content (v/v)		
Halothane	100 per cent	68.3 per cent
Ether		31.7 per cent
Boiling point °c	50.2	51.5

*A preliminary report of this work was presented by Dr Love at the Second World Congress of Anesthesiologists, Toronto, 1960

METHODS

The cases selected for the study were children five years old or over, about to undergo minor surgical procedures. They were divided into three groups according to the agent studied (TABLE 2). Statistical analysis revealed no significant difference in age or weight in the three groups.

Table 2

Details of subjects studied

AGENT	NO OF CASES	SEX	AVERAGE WEIGHT (KILOS)	AVERAGE AGE (YEARS)
Ether	10	4F/6M	28.2±2.40	9.0±0.33
Halothane	20	6F/14M	26.8±3.15	7.7±0.65
Azeotrope	20	4F/16M	26.8±2.45	8.5±0.68

Initially, several control measurements of respiratory data were made in each subject using a Wright Respirometer[8]. However, although every effort was made to allay apprehension the respiratory rate in each child was two or three times the expected normal; this was almost certainly the result of unaccustomed breathing into the meter. Thus, in the absence of reliable data, it was unfortunately necessary to use the Radford Nomogram[9].

Pre-anæsthetic medication was limited to atropine 0.65mg given subcutaneously thirty minutes before induction.

Anæsthesia was induced with a small amount of 2.5 per cent thiopentone (4mg/kg), followed by nitrous oxide 75 per cent, and oxygen 25 per cent and the volatile agent under investigation. Since at least fifteen minutes elapsed between the administration of thiopentone and the beginning of the study it was felt that this dose would not appreciably affect the results. An EMO, Fluotec and Azeotec vapouriser were used to study ether, halothane and the azeotrope respectively. A Ruben Valve was included to make a non-rebreathing system and the Wright ventilation meter was placed on the expiratory limb of this valve for the measurement of minute volume. The flow of fresh gases was such as to maintain a moderate and constant distension of the reservoir bag at the end of each expiration.

Arterial pressure was estimated by the auscultatory method (Riva Rocci) and this, together with the minute volume and respiratory rate, was recorded every three minutes. Mean arterial pressure was calculated as diastolic pressure plus one third of the pulse pressure. An ECG, Lead II, was used to monitor cardiac rhythm and provided an accurate measurement of heart rate.

In one subject not included in the series, consecutive periods of anæsthesia with halothane and azeotrope were obtained and end expired P_{CO_2} was measured with a Stanley Cox time phased carbon dioxide analyser (FIG. 1).

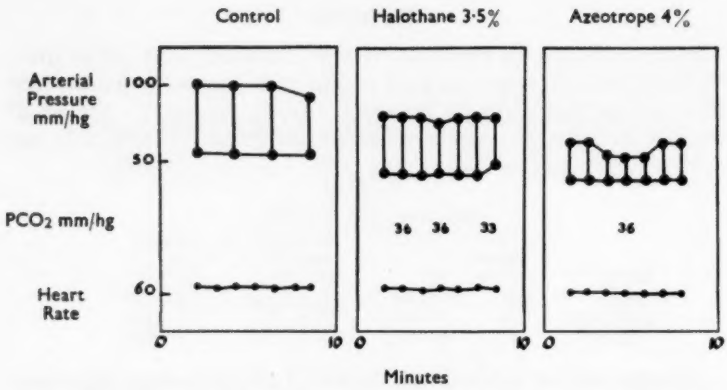

FIG. 1 Comparison of the effects of halothane and the azeotrope on arteria pressure and heart rate.

All the results presented are based on observations made over a period of ten to twenty minutes of stable anæsthesia prior to surgery.

Standard statistical methods were used to determine the significance of differences between observations, a difference being considered significant when the P value was less than 0.05.

RESULTS

Circulatory changes

In general the mean arterial pressure was maintained above the control level during ether anæsthesia. The inhalation of halothane failed to produce a significant alteration in pressure ($t=1.11$; $0.30 \quad P > 0.20$), and deviations greater than 10 per cent of the pre-anæsthetic figure were seldom observed. In contrast, consistent and profound reductions occurred when the azeotrope was administered, the degree of hypotension produced being highly significant ($t=6.41$; $P<0.001$). As expected, there was also a highly significant difference between the effects of halothane and the azeotrope on mean arterial pressure ($t=6.75$ $P<0.001$). These results are summarised in TABLE 3, and FIGS. 2 and 3.

Table 3

Summary of changes in mean arterial pressure

AGENT	VAPOUR CONCENTRATIONS VOLUMES PER CENT AVERAGE	RANGE	AVERAGE ABSOLUTE CHANGE IN MEAN ARTERIAL PRESSURE
Ether	7.6	5.0–10.0	+10.0±4.20
Halothane	1.46	0.6– 1.9	+ 3.7±1.87
Azeotrope	1.70	1.0– 4.0	−15.6±3.02

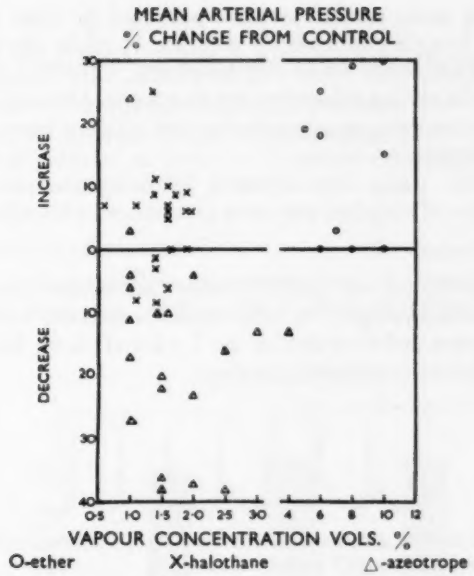

FIG. 2 Change in mean arterial pressure in relation to the concentration of ether, halothane and the azeotrope.

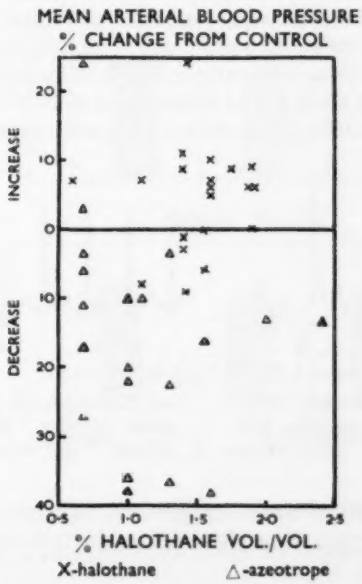

FIG. 3 Change in mean arterial pressure in relation to the concentration of halothane and azeotrope. (Expressed as per cent of halothane.)

The rise in mean arterial pressure produced by ether was due to increases in systolic and diastolic pressure. A slight rise in diastolic pressure was frequently noted with halothane, while both systolic and diastolic readings were reduced by the azeotrope. Although a decrease in pulse pressure was a constant finding with all three agents it was less marked with ether.

Tachycardia, which was expected following the pre-anæsthetic administration of atropine, was most pronounced with ether.

Cardiac rhythm

Normal sinus rhythm was usually present during ether anæsthesia and the only electrocardiographic abnormalities detected were S–T segment depression and inversion of the T wave (FIG. 4). Such changes may be attributed to sinus tachycardia.

FIG. 4 E.C.G. Lead II. 25mm/sec. Ether 10 per cent. Sinus tachycardia (160/min). S–T segment depression with T wave inversion.

In two cases bradycardia with a depressed and bifid P wave was recorded during induction with halothane although these changes reverted to normal without any reduction in vapour strength when anæsthesia became stable. In a third case pulsus bigeminus and multifocal ventricular extrasystoles occurred with tachypnœa and probable retention of carbon dioxide and disappeared when a reduction in the concentration of halothane permitted adequate ventilation to return (FIG. 5).

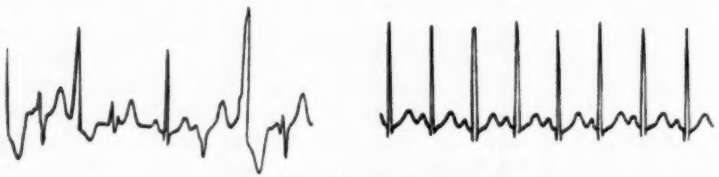

FIG. 5 E.C.G. Lead II. 25mm/sec.

(*a*) Halothane 2.5 per cent. Multifocal ventricular extrasystoles. Respiratory rate. 70/min. Tidal volume 110mls.

(*b*) Halothane 1.0 per cent. Normal sinus rhythm. Respiratory rate 54/min. Tidal volume 150mls.

With the exception of one instance, where nodal rhythm developed with induction, normal sinus rhythm was the rule during azeotrope anæsthesia (FIG. 6).

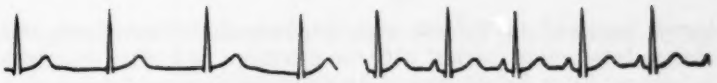

FIG. 6 E.C.G. Lead II. 25mm/sec.

(*a*) Azeotrope 1.5 per cent. Nodal rhythm during induction of anæsthesia.

(*b*) Azeotrope 1.5 per cent. Reversion to normal sinus rhythm.

Respiratory changes

The main respiratory effects are shown in FIG. 7. As mentioned the control data were obtained from the Radford Nomogram. All three

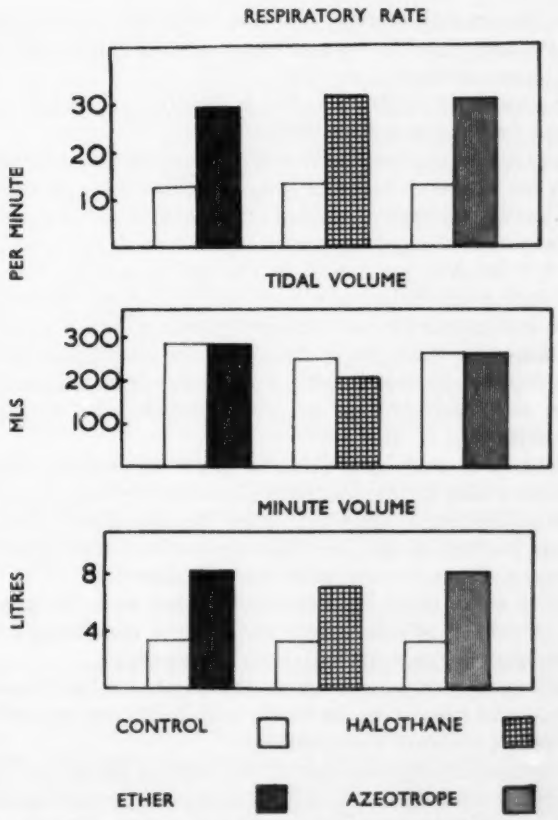

FIG. 7 The effects of ether, halothane and the azeotropic mixture on respiration.

agents increased the minute volume above the control level; this change being more marked with the azeotrope and ether than with halothane. Ether, halothane and the azeotrope each produced similar increases in the respiratory rate. Tidal volume was little altered by ether or the azeotrope, but was slightly reduced by halothane. This was reflected in the lower minute volume during halothane anæsthesia.

Pupils

The pupils were invariably widely dilated during ether anæsthesia and constricted, frequently to a pin point size, by halothane and the azeotropic mixture.

DISCUSSION

The most important finding of the work presented is the depressant effect of the azeotrope on the arterial pressure as compared with the action of its constituents.

It is of interest to consider possible explanations for what has been a consistent finding throughout the studies.

Price and his co-workers[10] measured the concentrations of catechol amines in the plasma of man and found that halothane, unlike ether, failed to increase sympatho-adrenal activity. This is in keeping with the clinical picture of vagal preponderance with bradycardia and nodal rhythm, hypotension, peripheral vasodilatation and constriction of the pupil during halothane anæsthesia. Similar changes were observed during the inhalation of the azeotropic mixture and so it seems reasonable to assume that this agent also elicited little sympathetic response. This suggests that the small concentrations of ether contained in the azeotrope were incapable of evoking compensatory increases in catechol amines.

The work of Brewster and his colleagues[11] on sympathectomised dogs and the studies on the isolated myocardium by Prime and Gray[12] and Price and Helrich[13] show that ether, even in subanæsthetic concentrations, is a myocardial depressant and will produce hypotension in the absence of an active sympatho-adrenal response.

Another possible cause for these findings may lie in the ganglionic blocking properties of halothane[14] and ether[15], combining to lower arterial pressure during anæsthesia with the azeotrope.

In addition to such considerations the combined local actions of halothane[16] and ether[17] on the blood vessel walls may contribute to the hypotensive action of the azeotrope.

One might assume that the absence of a marked fall in arterial pressure during the inhalation of halothane is due to hypercarbia counteracting its direct depressant effects on the circulation. However, this is unlikely to be the case with the azeotrope and FIG. 1 shows that such

changes in arterial pressure still occur when P_{CO_2} is within normal limits.

In the children studied, ventricular ectopic contractions were observed on only one occasion and this was during halothane anæsthesia when pulmonary ventilation was inadequate. Although further work is needed to determine whether or not the azeotrope protects against such irregularities in man some speculations may be made. The ability of small amounts of ether to abolish ventricular extrasystoles during cyclopropane anæsthesia is well known[18]. At first sight it would seem that ether, a sympathetic stimulant, should increase the tendency for adrenaline-hydrocarbon arrhythmias to develop. It may well be that ether exerts its influence by means of a vagal pulmo-cardiac reflex. Also, as the hypotensive action of the azeotrope indicates, there is probably little sympathetic activity due to the contained ether so arrhythmias should not be more common than with halothane. Indeed the fall in arterial pressure caused by the azeotrope may protect against the development of ventricular irregularities[19 20]. It has been shown that respiratory acidosis is less likely to occur with the azeotrope than with halothane, a further safeguard against the initiation of disorders of cardiac rhythm.

SUMMARY

The circulatory and respiratory effects of ether, halothane and the azeotropic mixture were studied in fifty children.

Arterial pressure was raised by ether, little altered by halothane and markedly reduced by the azeotrope. Possible explanations for this latter finding are discussed.

Halothane-ether azeotropic mixture had a stimulant effect on respiration as compared with halothane alone. Tidal volume was diminished by halothane, but unaltered by the azeotrope or ether. This resulted in a lower minute volume during halothane anæsthesia.

In theory ventricular arrhythmias would seem less likely to occur with the azeotrope than with halothane.

Acknowledgements

We wish to express our thanks to our surgical colleagues and the nursing staff of the Royal Belfast Hospital for Sick Children for their patient co-operation; to Dr J. W. Dundee for his helpful criticism and advice; to Dr R. S. J. Clarke, for his most willing assistance; to the Council of the Association of Anæsthetists of Great Britain and Ireland for the use of an Azeotec Vapouriser and to Dr H. G. Epstein, Nuffield Department of Anaesthetics, University of Oxford, for the special calibration of the Fluotec Vapouriser used in these studies. We are grateful to Mr John Beale, Pharmaceuticals Division, Imperial Chemical Industries Limited, for providing halothane (Fluothane) for this investigation.

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An investigation of new fluorine compounds in anæsthesia (3)

The anæsthetic properties of hexafluorobenzene

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The work described in '*Fluorine compounds in anæsthesia* (1)'[1] has continued. Investigations which have not yet been published have shown that fluorinated six-membered ring compounds are extremely toxic to mice. It was, therefore, considered at that time, that six-membered cyclic fluorinated compounds should, in future, be excluded as possible anæsthetic substances. One reason for this was that not only were these substances toxic, but there were no marked signs of anæsthesia preceding death with the compounds tested. Despite this fact, when the opportunity was presented, by Professor Stacey, of examining hexafluorobenzene C_6F_6 it was considered desirable to do so, because, although it is a six-membered ring compound, it is aromatic and hitherto, no experience has been obtained with aromatic compounds. This report describes the results of some preliminary experiments.

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HEXAFLUOROBENZENE

(*a*) *General*

The first detailed reference to hexafluorobenzene is given by McBee *et al.*[2] who state that Swartz[3] postulated that it was one of the products obtained by heating a platinum wire in an atmosphere of hexafluoroethane. No product was, however, isolated or identified by Swartz. McBee *et al.* were the first workers to isolate hexafluorobenzene, whilst the most recent reference to this substance is that of Forbes *et al.*[4].

(*b*) *Preparation*

Three methods of preparation of hexafluorobenzene appear in the literature, McBee *et al.*[2] made it from hexachlorobenzene, by treating it with bromine trifluoride, then reacting the intermediate product with antimony pentafluoride and finally obtaining hexafluorobenzene in poor yield by de-halogenation with zinc. Godsell *et al.*[5] prepared it by passing benzene vapour over cobaltic fluoride at 150°C to give nonafluoro*cyclo*hexane which was boiled with concentrated aqueous potassium hydroxide to give a 10 per cent conversion to hexafluorobenzene. Hellmann *et al.*[6] when searching for a method giving hexafluorobenzene more conveniently in better yield reverted to an earlier preparation of Desirant[7] suitably modified. This depends upon the pyrolysis of tribromofluoromethane (obtained from carbon tetrabromide and antimony pentafluoride in 60–70 per cent yield). The pyrolysis was best carried out at 540°–550°C in a platinum tube in nitrogen at 4.5 atmospheres pressure. This process gave a yield of 55 per cent on the tribromofluoromethane.

(*c*) *Properties*

Hexafluorobenzene has a boiling point of 80°C[5]. Earlier figures of 80.5°C[7] and 81–82°C[2] and also a freezing point −13 to −11°C[2] are probably less reliable because of doubts on the purity of the samples[5]. No reference appears in the literature either to the inflammability, or to the toxicity or otherwise of the substance. One would expect it to be stable to soda lime in view of the method of preparation with boiling alkali used by Godsell *et al.*[5]. Chemically, hexafluorobenzene is different from benzene itself since it is reactive to nucleophilic agents; for example, it gives pentafluoroaniline with sodamide[8] and pentafluoroanisole (pentafluoromethoxybenzene) and 1, 2, 4, 5-tetrafluoro-3, 6-dimethoxybenzene on boiling with methanolic sodium methoxide[5 8].

EXPERIMENTAL METHODS

(*a*) *Material*

We are indebted to Professor M. Stacey and his colleagues Professor

J. C. Tatlow and Dr Fuller for a first sample of 10g and to the Imperial Smelting Corporation, Avonmouth and Dr A. K. Barbour for a second sample of 50g. Both samples were purified by the respective donors and checked by us for purity by gas chromatography.

(*b*) *Mouse test apparatus*

Eight separate experiments were carried out involving fifteen mice in all. For the first three tests with the smaller sample, use was made of the simplified mouse test apparatus previously described[1]. With this apparatus one mouse is used per test in a small container of approximately 67ml capacity. An open circuit technique was used and two gas sample tubes were connected in series with the outlet from the mouse container. The contents were subsequently analysed. The substance was mixed with oxygen by means of a simple vaporiser which was, in effect, a test tube fitted with a wash-bottle head. Variations in gas flow rate, level of liquid in the tube and the temperature of the bath (if any) surrounding the tube were used to produce changes in the concentration of the vapour in oxygen.

The five experiments using the larger sample from Imperial Smelting were carried out in a more complicated apparatus. This comprises a desiccator of 1600ml capacity which holds up to six mice, the simple type of vaporiser as previously described, a circulating pump and a thermal conductivity analyser for monitoring the concentration of vapour administered. Open, semi-open, or closed circuit techniques could be employed with this apparatus. In all cases the mice were caused to breathe oxygen for at least ten minutes before receiving the hexafluorobenzene vapour, to remove nitrogen from the system as far as possible.

(*c*) *Recording results*

As in previous experiments, a tape recorder was used for recording events as they occurred. This was supplemented where desirable by written records and a typed copy of the experiment was kept by all taking part.

(*d*) *Analysis of samples and stability to soda lime*

The samples were analysed by the Bone and Wheeler method outlined previously[9]. Gas chromatography was used to test the stability to soda lime, the exposure being for three hours in a sealed tube at 70°C.

(*e*) *Inflammability tests*

The inflammability limits of hexafluorobenzene have not yet been determined and the tests reported here are only qualitative. In some cases outflow gas from the apparatus was collected over water in a

3 in × 1 in flat bottomed test tube and sealed during transit to a nearby Bunsen burner by a bark cork inserted while the tube was below the surface of the water. The flame was adjusted to about ½ in in height and the tube brought to it with the mouth inclined downwards. The cork was removed and in the same movement the mouth of the tube was passed through the flame. Ignition in proving trials using other inflammable vapours, was unmistakable and the negative results uniformly obtained with hexafluorobenzene are held to be genuine. In other trials a little of the liquid on a watch glass or in the bottom of a small beaker was flamed in the presence of air or oxygen.

RESULTS

(*a*) *Effects on mice* (*summary of experiments*)

1 0.2 per cent v/v hexafluorobenzene produced light anæsthesia in one mouse for thirty minutes.
2 1.6–2.4 per cent produced deep anæsthesia for thirty minutes in one mouse with complete, but slow, recovery.
3 4.4 per cent produced respiratory arrest followed by death in one mouse. Exposure was for twenty minutes.
4 1.25 per cent produced light anæsthesia for thirty minutes with rapid recovery (four mice used).
5 3.5 per cent produced very deep anæsthesia in two mice for thirty minutes, with slow recovery.
6 Six mice were lightly anæsthetised for twenty minutes on two consecutive days, with rapid recoveries.

The results show that hexafluorobenzene is an anæsthetic in relatively low concentrations.

(*b*) *Purity and stability to soda lime*

The results of gas chromatographic analysis of the hexafluorobenzene obtained from Birmingham before and after heating with soda lime were as follows:

Examination of hexafluorobenzene by gas chromatography

	CONSTITUENT	RETENTION TIME	PERCENTAGE OF 1 IN 2
Sample as received	1	80 secs	0.15
	2 (main peak)	160 secs	
Sample after heating at 70°C for 3 hours with soda lime in sealed tube	1	80 secs	0.05
	2 (main peak)	160 secs	

It will be seen that the sample was pure and unaffected by soda lime. The sample from Imperial Smelting contained one component only and was also unaffected by hot soda lime.

(*c*) *Inflammability tests*

One inflammability test was carried out during experiment (1) when the concentration was 0.2 per cent and as expected, no ignition took place.

Three inflammability tests were carried out during the course of experiment (2). In this experiment the hexafluorobenzene was surrounded by a water bath at 46–48°c and oxygen passed over the liquid. No ignition was obtained when samples were tested at six, twenty and twenty-seven minutes when the concentrations were approximately 2.4 per cent, 1.6 per cent and 1.6 per cent, respectively.

At the end of experiment (2) the unused hexafluorobenzene in the vaporiser was poured into a small watchglass and heated from below using a large flame. A yellow flash was seen indicating that the vapour had decomposed, but no combustion took place. On directing oxygen at the liquid, combustion took place with a smoky flame.

Further attempts to produce ignition with collected samples of oxygen and hexafluorobenzene were made during the course of experiment (4) at twenty-eight and twenty-nine minutes (concentration about 1.25 per cent). Again ignition did not take place.

A little hexafluorobenzene was left over from experiment (5) and the ignition test was repeated using a small beaker instead of a watchglass. Comparison was made with a sample of trichloroethylene. This was confirmed not to burn in air in any circumstances, but the warm liquid could be made to burn under an oxygen atmosphere. The same result was obtained with hexafluorobenzene.

The above results strongly indicate that hexafluorobenzene is non-inflammable in air and if inflammable in oxygen this might be under conditions not likely to be encountered in anæsthetic practice. In this respect hexafluorobenzene appears to resemble trichloroethylene.

Later investigations on the flash point of hexafluorobenzene show that the lower inflammability limit in oxygen is 5–6 per cent v/v.

DISCUSSION

It is of great interest that hexafluorobenzene has proved to be an anæsthetic for mice. This contrasts very markedly with the behaviour of all other six-membered ring compounds hitherto examined by the present team, which have always been either toxic or very toxic and devoid of any anæsthetic properties. During the course of the series of experiments reported here, only one mouse was lost. This was the mouse used in experiment (3) where a concentration of 4.4 per cent was administered. The anæsthetic level was so deep during fifteen minutes that respiration had either become very slow and shallow or had ceased altogether for periods of 4–5 secs. It is considered that this

mouse did not die because of any direct toxic action of hexafluorobenzene, but because of the anoxia and carbon dioxide accumulation resulting from very slow and irregular shallow respiration.

Another indication of the relative non-toxicity of hexafluorobenzene was given in experiment (5) where two mice were heavily anæsthetised for thirty minutes. Here again the anæsthesia was taken to the point of near respiratory arrest and although one mouse appeared to be dying shortly after the experiment it did in fact survive for a period of forty-eight hours.

During the course of these experiments a total of fifteen mice were anæsthetised and with one exception, all recovered. The ease of administration and safety of the drug were sufficiently great for it to be used in a demonstration to an Anæsthetists Meeting; this demonstration proceeded without any difficulty and two of the mice used were anæsthetised heavily and subsequently more lightly on four occasions spread over two days and their recovery was apparently complete.

The inflammability tests showed that hexafluorobenzene has a behaviour rather similar to that of trichloroethylene. It is non-inflammable in air, although under appropriate conditions its vapour can be caused to burn in oxygen. There was insufficient quantity of sample for quantitative estimates of the inflammability limits to be made.

The other results of interest with hexafluorobenzene were that the samples used were pure and did not react with soda lime when heated at 70°C for three hours in a sealed tube.

SUMMARY

A total of fifteen mice have been anæsthetised with hexafluorobenzene during eight separate exposure periods. The stability of the compound is high; it is unaffected by hot soda lime during exposure at 70°C for three hours and it is non-inflammable in air. Detailed inflammability limits in oxygen have not yet been determined, but from preliminary experiments it would appear to have inflammability characteristics similar to trichloroethylene, being non-inflammable in air, but with a lower limit in oxygen of 5–6 per cent v/v.

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Effect of posture on ventilation of patients anæsthetised with halothane

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It has long been realised that changes in posture may influence the ventilation of the anæsthetised subject. Few measurements, however, have been made. Reference is frequently made to the work of Case and Stiles[1], but their paper refers to changes in vital capacity of conscious subjects when changed from the sitting position to various other positions. There is not necessarily any correlation between changes in vital capacity and ventilation, unless ventilation is limited by vital capacity which is seldom the case.

The lack of data has prompted us to undertake the present study in which we have investigated the effect of posture upon the ventilation of human subjects anæsthetised for routine surgical procedures.

METHOD

Anæsthesia

The twenty patients used for this study were drawn from routine operating lists. Their ages ranged from twenty-one to seventy-two years. None showed clinical or radiological evidence of cardiac or pulmonary disease. The surgery did not require the use of muscle relaxants; the commonest procedures were cystoscopy and inguinal herniorrhaphy.

Premedication was with pethidine and atropine in most cases. Three received promethazine as well, while four were premedicated with papaveretum and hyoscine.

Anæsthesia was induced with thiopentone (200–400mg).

This was followed by suxamethonium (50mg) and a cuffed endotracheal tube was then passed. Anæsthesia was maintained with nitrous

oxide and oxygen (5:2 l) with halothane (1–2 per cent) added by means of a 'Fluotec' vaporiser in a Magill circuit. This technique permitted the maintenance of a reasonably constant depth of anæsthesia and avoided the changes in ventilation due to intermittent supplementation of nitrous oxide with pethidine, thiopentone and muscle relaxants. All studies were carried out immediately after the end of the surgical procedure, thus avoiding changes in ventilation due to surgical stimulation.

Measurement of ventilation

As the operation drew to a close, the anæsthetic gas circuit was replaced by a non-rebreathing system with a continuous flow spirometer[2], the composition of the fresh gas supply being unchanged. This provided a continuous tracing of respiration with an accuracy of better than 2 per cent of the observed tidal volume. Control measurements were made with the patient in the supine position. The position was then changed to one of those described below without interruption of the spirometer trace – the change being made as rapidly as possible. Changes in ventilation were calculated as a percentage of the mean of the levels of ventilation before and after positioning. The new level of ventilation was established as soon as the new position was attained and there were no consistent changes over the following few minutes. It, therefore, appeared unnecessary to maintain the position for a longer time: indeed, doing so would have made comparison difficult because of the long term variations in ventilation which are a feature of anæsthesia with spontaneous respiration. Changes into the prone and the lateral positions were sometimes complicated by coughing or breath-holding lasting for a few seconds.

Changes in expiratory lung volumes were derived directly from the spirometer trace.

The positions

The patients were moved directly from the supine position into certain positions which we have called Primary Positions:

Trendelenburg 30° head down, with slipping prevented by pelvic rests.

Gall bladder supine, with the gall bladder rest raised 4in (10cm) at the level of 8th–10th thoracic vertebra.

Lateral table level, knees and hips slightly flexed, rotation prevented at shoulders and pelvis.

Reverse Trendelenburg 30° head up, slipping prevented by foot plate or pelvic rest.

Lithotomy table level, hips and knees flexed so that knees were just clear of the thorax; lumbar vertebræ fully flexed.

Prone table level, head turned to one side; pelvis and shoulders unsupported.

The attainment of two further positions required that the patient be first moved into one of the Primary Positions; these we have called Secondary Positions.

Jack-knife patient placed prone for one to two minutes, then the table broken at the level of the second lumbar vertebræ to 110°.

Kidney patient placed in the lateral position for one to two minutes, then the table broken as for the jack-knife position, but to 135°.

Ventilation in each secondary position was compared with that in the primary position from which it was derived, and also with that in the supine control position by taking the product of the changes occurring in the two stages.

RESULTS

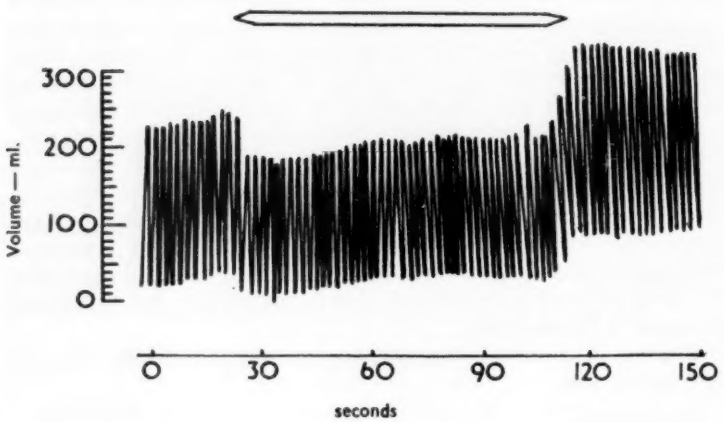

FIG. 1 Typical spirometer record obtained during positioning in the Trendelenburg position (between 30 and 120 seconds).

FIG. 1 shows a typical record which demonstrates the absence of any further changes of ventilation after the position was established. The functional residual capacity and tidal volume were usually altered, but, in contrast, the respiratory frequency was seldom significantly changed.

The observed changes in tidal volume and respiratory frequency are shown in FIG. 2. Mean changes in minute volume are more easily seen in TABLE I which also shows the changes in functional residual capacity. All changes in minute volume appear significant except in the lithotomy and prone positions. The changes in respiratory frequency are random and not significant.

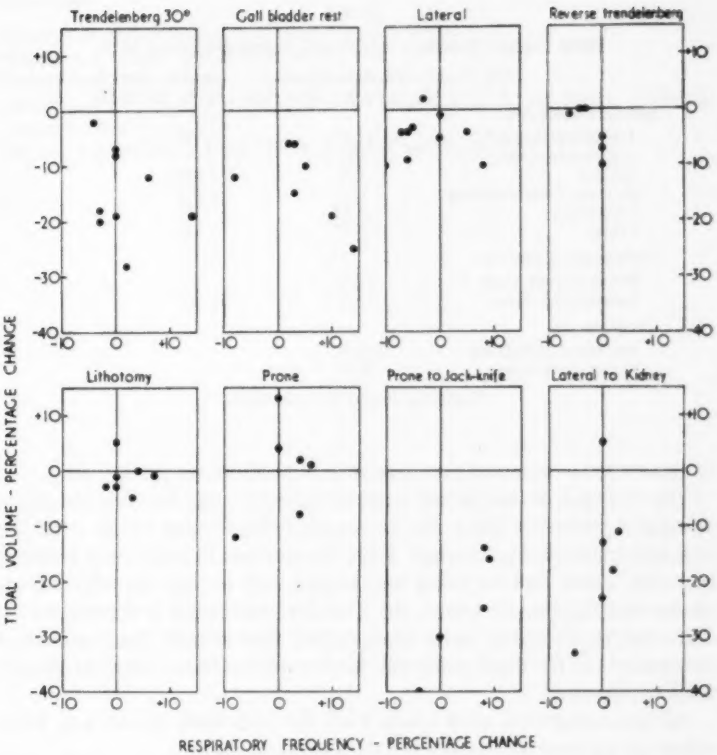

FIG. 2 Percentages changes of respiratory frequency and tidal volume on assuming the positions described in the text.

DISCUSSION

It will be seen that the minute volume of ventilation was considerably reduced in the kidney, prone jack-knife and Trendelenburg positions. The reduction often appeared sufficient to require artificial ventilation. There was an appreciable but lesser reduction in the gall-bladder, lateral and reverse Trendelenburg positions. The lack of significant change in the prone and lithotomy positions contrasts with the considerable reduction in vital capacity in these positions[1].

Our results for the Trendelenburg, lateral and lithotomy positions agree well with a study reported by Jones and Jacoby[3]. In other positions, however, the agreement is not good. Comparison is difficult, since these authors give few details of the positions studied and no information as to the number of patients investigated.

Table I

Mean changes in minute volume and resting expiratory level

	MINUTE VOLUME CHANGES PER CENT	RESTING EXPIRATORY LEVEL ML (BTPS)
PRIMARY POSITIONS		
Trendelenburg 30°	−13.9	− 95
Gall bladder rest	− 9.6	
Lateral	− 8.7	+300
Reverse Trendelenburg	− 6.2	+213
Lithotomy	+ 0.3*	− 67
Prone	+ 1.5*	+ 70
SECONDARY POSITIONS		
Prone to jack-knife	−21.2	
Lateral to kidney	−15.5	+ 10
Total changes		
Supine to jack-knife	−20.0	
Supine to kidney	−22.8	

*Indicates results not significant

Others have reviewed the literature, but offered no further data.

The changes in functional residual capacity may be of some slight clinical significance since the anatomical dead space varies with the end-inspiratory lung volume[4]. Thus, an increase in functional residual capacity, other factors being unchanged, will reduce the effective alveolar ventilation. However, the alveolar ventilation is dependent on the total physiological dead space rather than merely the anatomical component of the dead space and the former has been shown to change with ventilation[5].

All measurements were made with the abdomen closed and have taken no account of the effect of packs and retractors.

It was not our intention to study circulatory phenomena but routine measurements of blood pressure by the Riva-Rocci method failed to demonstrate any changes greater than 10mm/Hg which could be attributed to changes in posture.

SUMMARY

The ventilatory effects of changes of posture have been studied in anæsthetised man. The kidney, prone jack-knife and Trendelenburg positions caused 14–24 per cent reduction in ventilation. Lateral, reverse Trendelenburg and gall bladder positions caused 6–10 per cent reduction while the lithotomy and prone positions caused no significant changes.

Acknowledgement

We are indebted to Mr R. Shackman and Mr A. K. Monro for permission to study patients under their care.

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The safety of neostigmine

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Cardiac arrest has been reported[1 2 3 4] following the use of neostigmine to reverse muscular relaxation obtained by d-tubocurarine chloride or gallamine in anæsthetised patients. For this reason many anæsthetists are hesitant to reverse curarisation in this way. Using a combination of very light general anæsthesia with nitrous oxide and oxygen, full relaxation with d-tubocurarine, and deliberate hyperventilation, the immediate serious cardiac effects have not been seen by the writers, although neostigmine has always been injected in a dose of 5.0mg at an interval after atropine 1.2mg.

It seemed possible that this apparent freedom from serious cardiac disturbance might be associated with the state of respiratory alkalosis which has been shown to occur when this anæsthetic technique is employed[5]. It is well known that high blood tensions of carbon dioxide may be associated with electrocardiographic evidence of cardiac disturbance[6 7] and it was thought that this might bear some relation to the more serious effects of neostigmine.

It was decided to observe the immediate cardiac effects of neostigmine by means of electrocardiography in a series of healthy patients undergoing routine surgical procedures and to attempt to correlate the findings with changes in acid-base balance.

METHOD

There were twenty-six adult patients in the series. The results in two cases were discarded because of technical difficulties. The remaining twenty-four patients, of whom nine were males, showed normal electrocardiograph tracings before operation and no evidence of cardiac disease on routine clinical examination. Studies before operation showed that in no patient was there any metabolic disturbance in acid-base balance as estimated by the 'Standard Bicarbonate' method of

Jørgensen and Astrup[8]. The operations performed are listed in TABLE 1.

Table 1

Gall bladder and bile ducts	11
Hernia	6
Appendix	5
Ureterolithotomy	1
Vagotomy	1
TOTAL	24

ANÆSTHETIC TECHNIQUE

The premedication consisted of morphine 10mg and atropine sulphate 0.6mg in all patients. Thiopentone in 2.5 per cent solution was used to induce anæsthesia in twenty-three patients. In one patient, pre-oxygenation preceded nitrous oxide and oxygen and thiopentone was omitted. In all patients endotracheal intubation was performed and for this, and for subsequent muscular relaxation, d-tubocurarine chloride was used in doses sufficient to allow full manual control of respiration. Anæsthesia was maintained using nitrous oxide and oxygen only. No other supplementary agent, intravenous or inhalational, was employed. A Boyle Mk II or a Coxeter-Mushin absorption system was employed.

ESTIMATION OF RESPIRATORY ACID-BASE CHANGES

Following the method of Brooks and Wynn[9] arterialised venous blood was withdrawn into heparinised syringes, the dead space of which had been filled with neutralised mineral oil. The syringes were sealed with soldered needle hubs and were immediately placed in iced water in a vacuum flask. Samples were withdrawn at fifteen minute intervals during the course of each operation and again at the time of administration of neostigmine.

The blood pH was measured anærobically at 38° C in a micro-flow glass electrode system, using a Vibron Model 33B pH Unit. The electrode cell was standardised with a phosphate buffer using the buffer scale interpolated by Mattock[10]. The normal range of arterialised venous blood obtained with this method was between 7.40 and 7.44. Carbon dioxide blood tensions were measured by a modification of the Astrup interpolation method[11 12]. All estimations were carried out within two hours of obtaining samples.

ELECTROCARDIOGRAPH MONITORING

Before operation the patient was connected to a direct writing electro-

cardiograph. Lead II was used throughout this study. Records were obtained before anæsthesia, during operation at intervals and continuously following the administration of atropine and neostigmine. No electrical or surgical stimulus was permitted to interfere with the electrocardiographic recording following the injection of neostigmine. The period of observation was limited by the return of consciousness and of spontaneous muscle activity. Needle electrodes were used throughout this study and the millivolt-calibration was checked before and after the end of each recording.

USE OF NEOSTIGMINE

The mode of respiratory control during the maintenance of and termination of anæsthesia was carried out in three different ways, In the first group of seven patients deliberate hyperventilation was practised during maintenance and was continued with absorption of carbon dioxide until full reversal was obtained. In the second group of six patients deliberate hyperventilation was also carried out during maintenance, but during reversal spontaneous respiration was permitted. Carbon dioxide absorption was maintained. In the third group of eleven patients respiration was controlled, but deliberate hyperventilation was not practised. During reversal spontaneous respiration was allowed and carbon dioxide absorption was discontinued. Obvious hypoxia was carefully avoided by assistance of ventilation. Atropine 1.2mg was injected intravenously when reversal was required and this was followed after a period of approximately five minutes by neostigmine 5.0mg.

RESULTS

In the first group the average blood pH during operation lay within the range 7.50 to 7.67 and the average pCO_2 lay between 18.5 and 30.0mm/Hg.

At the time of administration of neostigmine the blood pH lay between 7.50 and 7.68 (FIG. 1). The pCO_2 levels were within the range of 18.0 and 29.4mm/Hg. In this group, changes in the electrocardiogram were confined to bradycardia, which was noted in three patients in this group of seven. FIG. 2 is a specimen tracing showing an absence of change.

In the second group the average blood pH during anæsthesia was within the range 7.47 to 7.55 and average pCO_2 was within the range 22 and 37mm/Hg. At the time neostigmine was administered, however, the blood pH range had fallen to 7.41–7.49 (FIG. 1) and pCO_2 range to 25 and 40mm/Hg.

Electrocardiograph changes were noted in two patients who showed

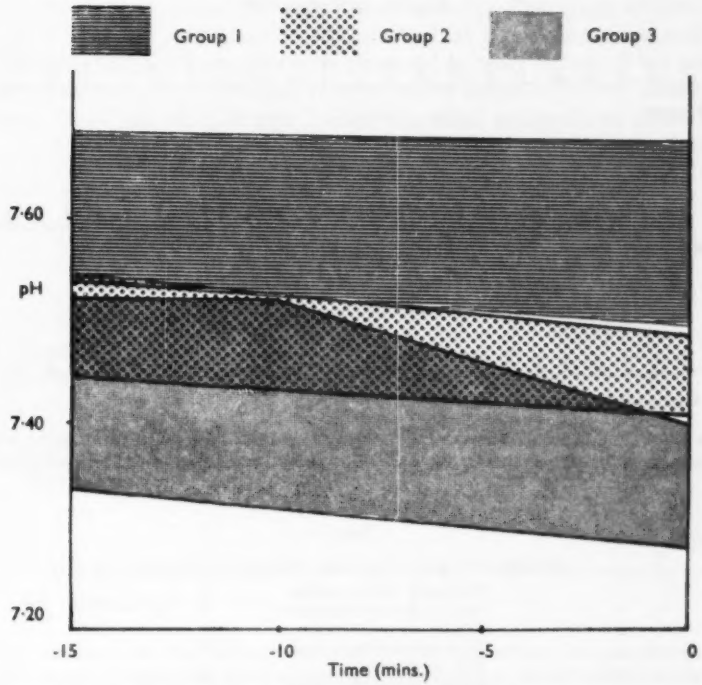


FIG. 1 Showing the range of blood pH during the last fifteen minutes of anæsthesia, for each group of patients. Neostigmine 5mg was given at '0'.

Group 1 patients who were hyperventilated with carbon dioxide absorption continuously, can be seen to maintain a marked degree of alkalosis at the time of administration of neostigmine.

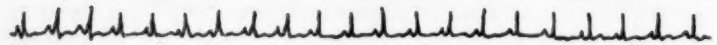
Group 2 patients who were hyperventilated during maintenance, but allowed spontaneous respiration with carbon dioxide absorption during reversal can be seen to still alkalotic at the time of administration of neostigmine.

Group 3 patients in whom deliberate hyperventilation was not performed and spontaneous respiration without carbon dioxide absorption allowed during reversal can be seen to be either at normal blood pH levels or acidotic at the time of administration of neostigmine.

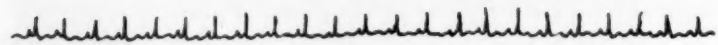
a slight diminution in voltage. Bradycardia was observed in only two instances.

In the third group during operation the average blood pH varied from 7.34 to 7.53 and average pCO_2 from 28 to 48mm/Hg. At the time of neostigmine administration the average blood pH varied from 7.28

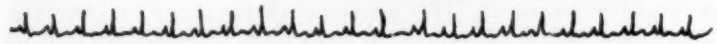
Case 20. Final pH_{38} 7·54



Normal



After atropine



After neostigmine

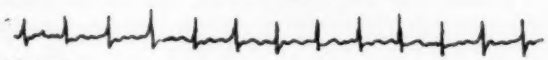
FIG. 2 ECG traces before anæsthesia, after the administration of atropine and after neostigmine. In this Group I alkalotic patient no changes were demonstrated.

Table 2

Electrocardiographic changes observed in Group III

Transient cardiac arrest	1
Ventricular extrasystoles	3
Atrioventricular block	1
Auricular extrasystoles	1
S–T depression	2
Gross voltage reduction	7
Changes in P wave	2
Diphasic T wave	1

Case 6 Final pH_{38} 7·38



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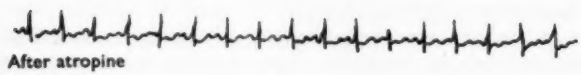

After atropine

After neostigmine

FIG. 3 ECG trace of a Group 3 patient demonstrating a transient cardiac arrest

to 7.40 (FIG. 1) and pCO_2 from 33 to 59mm/Hg. This range of blood pH is acidotic according to the method of estimation employed. Electrocardiograph changes were observed in every patient. They are listed in TABLE 2. Transient cardiac arrest occurred in one patient (FIG. 3). FIGS. 4, 5, 6 and 7 illustrate other examples of electro-

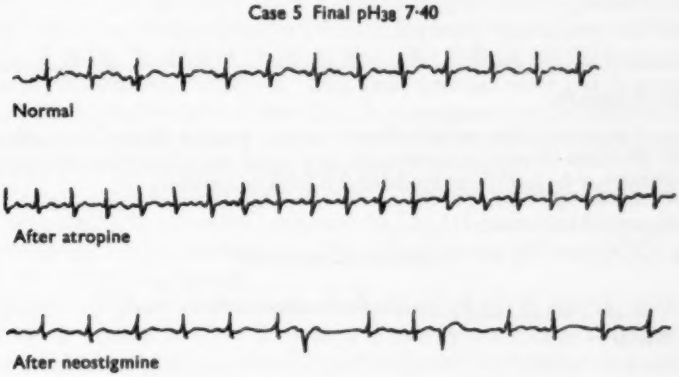

FIG. 4 ECG trace of a Group 3 patient who was mildly acidotic. The sample trace demonstrates ventricular extra systoles after neostigmine, which continued until muscular movement and return of consciousness prevented further ECG recording.

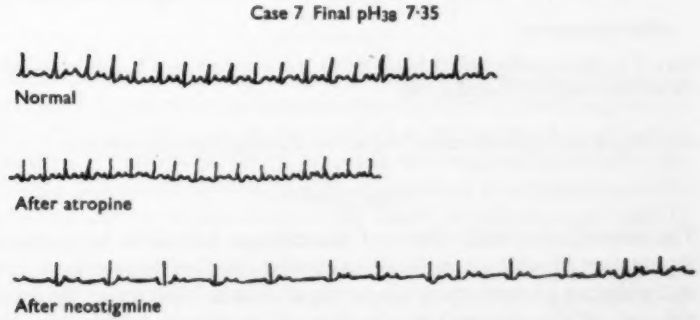

FIG. 5 ECG trace of an acidotic Group 3 patient demonstrating a partial second degree A–V block.

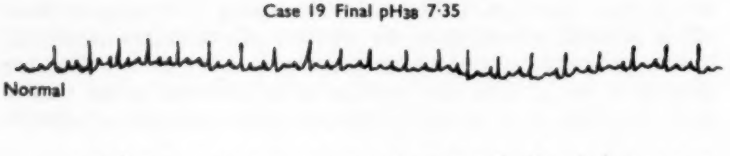

FIG. 6 ECG trace of an acidotic Group 3 patient showing diminution in voltage after the administration of neostigmine. The use of needle electrodes and careful calibration of the instrument precludes this being an artefact.

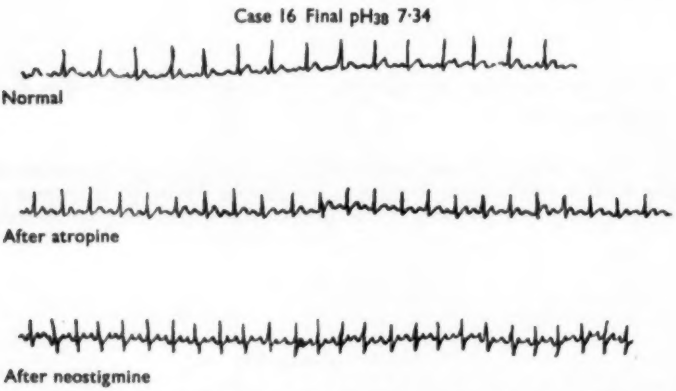

FIG. 7 Acidotic patient of Group 3. ECG demonstrates an S.T. depression after the administration of neostigmine.

cardiographic changes observed in the third group of patients.

DISCUSSION

The immediate cardiac effects of neostigmine appear to be related to the manner in which ventilation is carried out during anæsthesia and to the degree of respiratory change in acid-base balance (as shown by pH and pCO_2 changes) at the time of neostigmine injection. The patients in group II in whom hyperventilation was carried out as a deliberate policy suffered a relatively small change in pH and pCO_2

during the period in which spontaneous respiratory activity was looked for prior to neostigmine administration (FIG. 1 group 2). In these the electrocardiographic changes were not marked. However, with the exception of bradycardia these changes were completely absent in patients in whom ventilation and absorption were continued (FIG. 1 group 1).

On the other hand when controlled respiration had not been carried to the point of deliberate hyperventilation, it was found that the pH and pCO_2 changes occurring during the period when spontaneous respiration was returning indicated some degree of respiratory acidosis (FIG. 1 group 3). It was in this group that serious disturbances in cardiac rhythm were observed. That these changes were not due primarily to the build up of carbon dioxide is suggested by the absence of electrocardiographic changes prior to the administration of neostigmine in patients who were not deliberately hyperventilated.

It seems probable that the immediate effects of neostigmine on the healthy heart after atropine, as judged by Lead II electrocardiographic monitoring, are dependent upon the state of blood pH and pCO_2 at the time when it is injected.

If this is so then certain recommendations appear to follow. While the use of a pause to allow the return of spontaneous respiration may be thought desirable, this should not be allowed to continue to a state of respiratory acidosis. Churchill-Davidson and Wise[13] have given their opinion that the practice of administering anticholinesterase drugs only after the first signs of the return of respiration is unjustifiably cautious. The findings reported here strongly support this view. Ventilation and absorption of carbon dioxide should be continued during the period of reversal. The use of deliberate hyperventilation appears to protect the healthy heart against the potentially harmful effects of neostigmine.

SUMMARY

An electrocardiographic investigation was carried out in twenty-four healthy adult patients anæsthetised using thiopentone (with one exception) nitrous oxide and oxygen with d-tubocurarine for muscular relaxation. The purpose was to ascertain the relationship between the acid-base balance at the time of administration of neostigmine and the immediate effects of this drug on the heart as judged from Lead II ECG records.

In a group of seven patients who were hyperventilated throughout anæsthesia and in whom curarisation was reversed during continued carbon dioxide absorption, bradycardia was observed in three patients.

Six patients were hyperventilated during maintenance of anæsthesia and allowed to breathe spontaneously with continued carbon dioxide

absorption during the reversal period. In two patients voltage diminution was observed and bradycardia was noted in two other patients.

In eleven patients respiration was controlled, but hyperventilation was not deliberately carried out. During reversal spontaneous respiration was allowed without carbon dioxide absorption. Electrocardiographic changes were noted in all cases. Transient cardiac arrest occurred in one patient.

It is suggested that in healthy patients the heart is protected from the effects of neostigmine by a respiratory alkalosis.

Acknowledgement

We wish to thank Dr L. H. Harris who assisted us in the interpretation of the electrocardiograms.

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Anatomy for anæsthetists

(2) The anterior abdominal wall

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Illustrated by

MARGARET C. McLARTY

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The detailed anatomy of the abdominal wall is essential knowledge to any anæsthetist who proposes to perform safe and effective regional blocks in this area.

(A) LANDMARKS

A few fixed points enable surface markings to be correlated with deeper structures:

(1) The *xiphoid* lies opposite the body of the ninth thoracic vertebra.

(2) The *costal margin* extends downwards and outwards from the xiphoid to the lower margin of the tenth rib, beyond which the eleventh and twelfth ribs are not long enough to extend. This lower border of the thoracic cage (the *subcostal plane*) is at the level of the third lumbar vertebra.

(3) The *transpyloric plane*, which passes through the first lumbar vertebra, lies half-way between the suprasternal notch and the pubis; it is good enough to regard this level as a hand's breadth below the xiphoid.

(4) A line joining the iliac crests passes through the fourth lumbar vertebra; it is thus a convenient safety level for lumbar puncture, since the spinal cord terminates at the L1/L2 junction.

(5) The umbilicus lies at the level of the L3/L4 interspace. It is an inconstant landmark and is lower in the child, in the obese and in the pregnant.

(B) FASCIA

There is no deep fascia over the trunk – its presence would render deep breathing or abdominal distension impossible. The superficial fascia, or fat, over the lower abdomen is, however, more fibrous on its deep aspect. It is customary to term the more superficial fatty subcutaneous tissue 'Camper's fascia' and the deeper fibrous tissue 'Scarpa's fascia'. There is, in fact, no true anatomical differentiation into these two separate layers[1].

(C) MUSCLES (FIGS. 1–3)

Rectus abdominis has a 1in wide origin from the pubic crest and symphysis and a 3in wide insertion into the fifth, sixth and seventh costal cartilages. The segmental developmental origin of this muscle is commemorated by three fibrous intersections which are situated at the level of the umbilicus, the level of the xiphoid and half-way between the two; a fourth intersection is sometimes found below the umbilicus. These intersections are present only on the anterior aspect of the muscle where each adheres to the anterior rectus sheath. Injected local anæsthetic is thus prevented from free dissemination throughout the anterior compartment of the rectus sheath although it will spread without hindrance in the posterior sheath.

Pyramidalis is a small, inconstant, triangular muscle which arises from the pubis, lies in front of the rectus and is inserted into the linea alba.

The *rectus sheath*. The rectus lies within a sheath formed, in the main, by a split in the internal oblique aponeurosis. Posteriorly this sheath is reinforced by the aponeurosis of transversus abdominis and anteriorly by that of the external oblique.

This basic arrangement is altered at both extremities: above the costal margin, the rectus lies directly on the costal cartilages. The anterior sheath here consists only of the external oblique aponeurosis, simply because neither internal oblique or transversus extend above the rib margin. For 2 or 3in below the costal margin, transversus abdominis remains muscular almost to the midline; these muscle fibres can be seen distinctly in the posterior wall of the upper part of the rectus sheath. Below a level half-way between the umbilicus and pubis, demarcated by the arcuate line of Douglas, the aponeuroses of all three lateral muscles pass in front of rectus. Here, then, posteriorly the rectus rests against transversalis fascia, extraperitoneal fat and peritoneum. The posterior rectus sheath can thus be said to be, from above downwards: (I) Cartilaginous (above the costal margin). (II) Muscular (where the muscle fibres of transversus obtrude into the posterior sheath). (III) Aponeurotic (the main bulk of the sheath). (IV) Areolar (below the arcuate line of Douglas).

The resistance of the tough fascia of the anterior wall of the rectus sheath is easily appreciated throughout its extent by the anæsthetist's needle. The posterior wall is less readily defined both above the pubis, where the sheath is deficient, and also high in the epigastrium, where it is softly muscular and not a tough, fibrous aponeurosis[2].

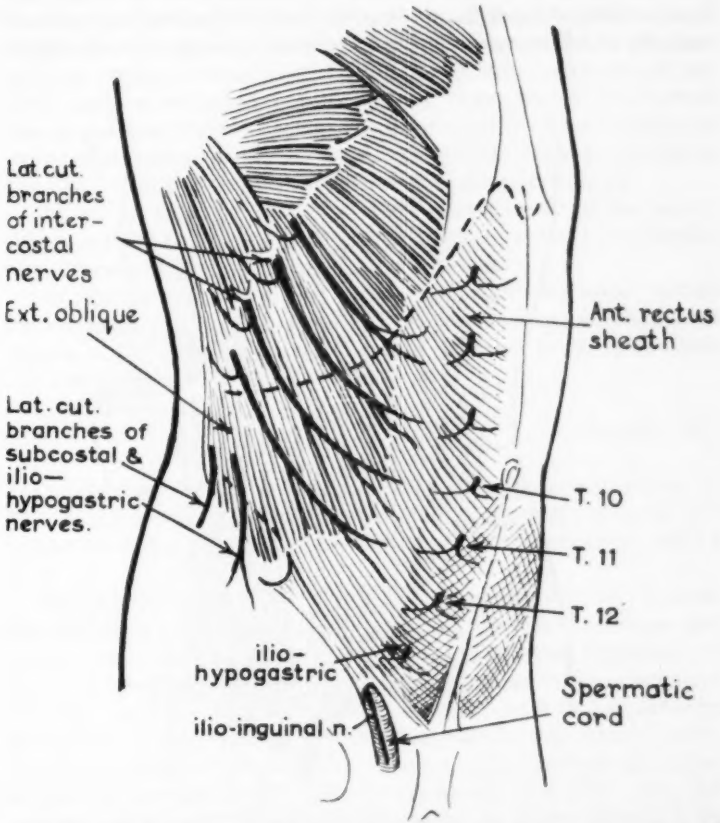

FIG. 1 View of abdominal wall after removing skin and superficial fascia. The lateral cutaneous and terminal branches of the nerves of the abdominal wall are demonstrated.

The aponeuroses which form the rectus sheath fuse from pubis to xiphoid in the almost avascular mid-line *linea alba*. This is narrow and quite difficult to define in the lower abdomen, but broadens out considerably above the umbilicus.

The three lateral muscles of the abdominal wall fill the space between the rectus in front, the lumbar muscles behind, the costal margin above and the iliac crest below. Their medial extensions constitute the rectus sheath, as described above, and then fuse into the linea alba in the mid-line.

Above the level of the iliac crest, the fibres of *external oblique* pass downwards and medially, those of *internal oblique* pass upwards and medially and those of *transversus abdominis* pass transversely. Below

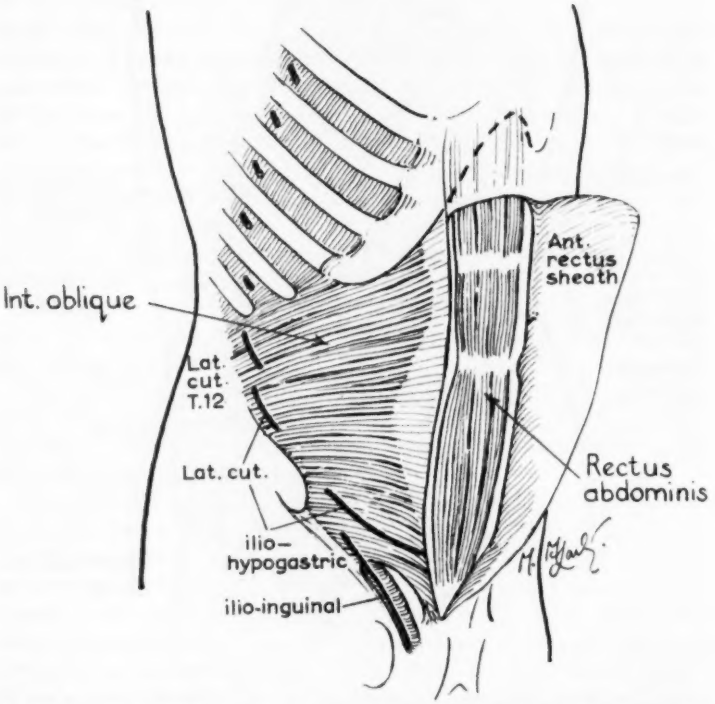

FIG. 2 The anterior rectus sheath has been opened, the external oblique removed, and the chest wall dissected down to the internal intercostal muscles.

this level, all the muscles are aponeurotic and all their fibres pass downwards and medially in the formation of the inguinal canal.

The muscles of the abdominal wall are accessory muscles of respiration which come into play in forcible expiration and coughing; they

act by raising the intra-abdominal pressure and also by drawing the lower ribs downwards and medially. Electromyographic studies have shown that the abdominal muscles are not used in inspiration[3].

(D) BLOOD SUPPLY

There is a rich blood supply to the abdominal wall; its details are unimportant to the anæsthetist except for the position of the inferior and superior epigastric vessels, which lie in the posterior rectus sheath and which may be wounded in performing a rectus block. The surface marking of these vessels is a line which curves gently from the femoral pulse at the groin to a point one finger's breadth lateral to the umbilicus and which runs thence vertically upwards to the costal margin.

The *inferior epigastric artery* is derived from the external iliac artery, skirts medially to the internal inguinal ring and enters the posterior rectus sheath beneath the arcuate line of Douglas.

The *superior epigastric artery* is smaller; it enters the upper part of the rectus sheath behind the seventh costal cartilage as a terminal branch of the internal mammary artery, runs vertically downwards and anastomoses with the inferior artery.

(E) NERVE SUPPLY

The abdominal wall is innervated by the anterior primary rami of T7–L1. Its segmental cutaneous supply is readily mapped out if it is remembered that T7 supplies the xiphoid, T10 the umbilicus and L1 the groin.

The intercostal nerves T7 to T11 and the subcostal nerve T12 enter the abdominal wall between the interdigitations of the diaphragm and transversus abdominis (FIG. 3). The intercostal nerves maintain the same relationship to the muscles of the abdominal wall as they have with the intercostal muscles. In their thoracic course they lie between the second and third layers of intercostal muscles (the internal intercostals and innermost intercostals); in their progress between the lateral abdominal muscles they lie between the second and third layer, the internal oblique and transversus abdominis, as shown in FIG. 4. In this plane, the nerves are conducted medially behind the rectus which they then pierce to supply the overlying skin.

In contrast, the first lumbar nerve divides in front of quadratus lumborum into the ilio-hypogastric and ilio-inguinal nerves which penetrate the transversus abdominis to lie between transversus and internal oblique.

The *ilio-hypogastric nerve* (FIGS. 2 and 3) pierces the internal oblique immediately above and in front of the anterior superior iliac spine,

runs deep to the external oblique, just superior to the inguinal canal, and ends by supplying the suprapubic skin.

The *ilio-inguinal nerve* (FIGS. 1–3) also pierces the internal oblique and then traverses the inguinal canal in front of the spermatic cord. It emerges either through the external ring itself or through the adjacent

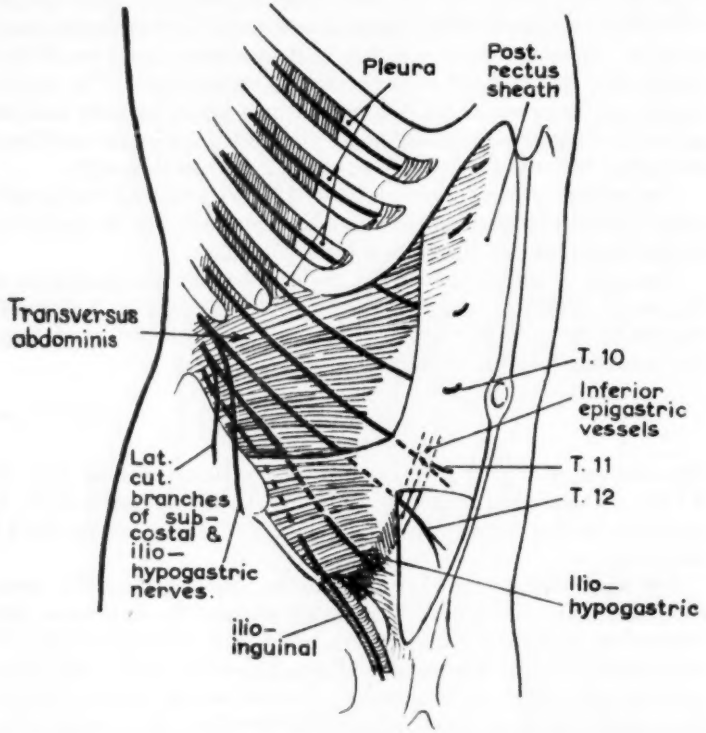

FIG. 3 Rectus abdominis has been removed revealing the posterior rectus sheath. In the upper abdomen a window has been cut out of the internal oblique revealing the lower intercostal, subcostal and L I nerves lying on the transversus abdominis. The innermost intercostal muscle layer is shown in each intercostal space.

external oblique aponeurosis to supply the skin of the scrotum (or labium majus) together with adjacent upper thigh.

Each nerve, apart from the ilio-inguinal, gives off a lateral cutaneous branch in the mid-axillary line. These branches from the intercostal nerves T7 to T11 divide into an anterior and posterior branch supply-

ing the skin from the lateral edge of rectus in front to the erector spinæ behind. The lateral cutaneous branches of both the subcostal (T12) and ilio-hypogastric nerve do not divide, but run downwards to supply the skin over the upper lateral aspect of the buttock (FIGS. 1–3).

Each nerve T7 to T12 gives off a small collateral branch which runs parallel with it; by analogy the ilio-inguinal nerve is to be regarded as the collateral branch of the ilio-hypogastric nerve and hence has no lateral cutaneous branch.

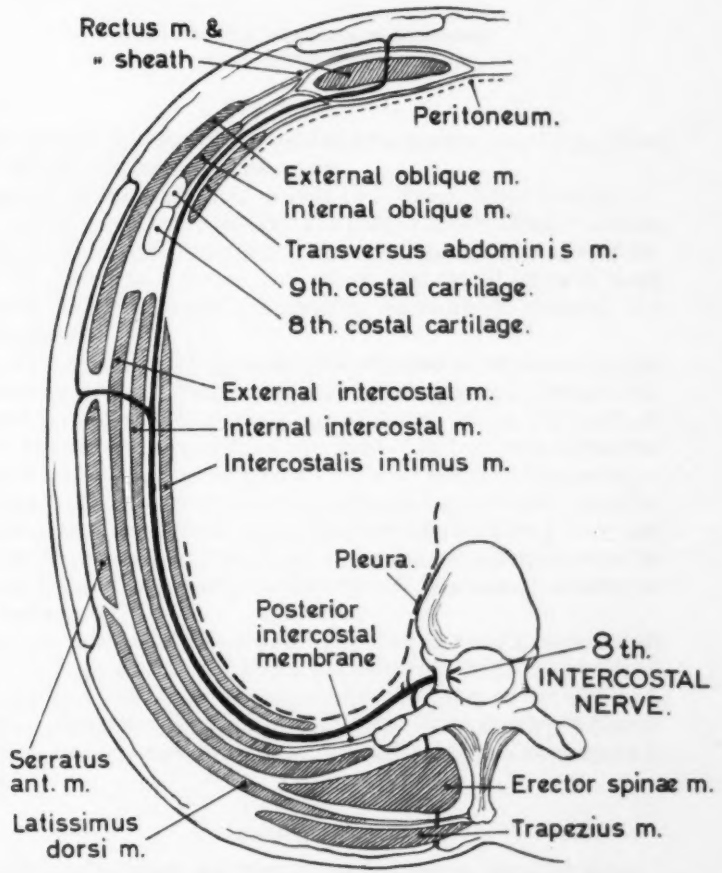

FIG. 4 A diagrammatic section of the distribution of the eighth intercostal nerve – note its relationship to the body-wall muscle layers is similar in its thoracic and abdominal course.

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The toxicity of lignocaine 2 per cent

A case report

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The purpose of this report is to condemn the routine use of lignocaine 2 per cent as a regional analgesic agent.

Lignocaine was synthesised in 1943 by Löfgren[1,2] and Lundqvist[2]. Early clinical reports were published by Bremer *et al.*[3], Gordh[4], Dabbs and Southworth[5] and Carnegie and Hewer[6]. General acceptance of the drug was rapid and it is now very widely used for all forms of local analgesia, including topical, infiltration, nerve-block, epidural and spinal analgesia.

At first the toxicity of lignocaine was reported to be similar to that of procaine and Goldberg[7,8], Gordh[4], Carnegie and Hewer[6] and Hunter[9] all agreed that a total dose of 1g was within the limits of safety. Possibly because of this assessment of its toxicity and because procaine was commonly used as a 2 per cent solution, lignocaine, a much more potent drug, came to be used in similar strength for routine infiltration and nerve-block analgesia. However, Goldberg[7,8,10] had stated that in concentrations of 1 per cent and over, lignocaine was the more toxic agent and Hunter[9] estimated that lignocaine was twice as toxic as procaine.

The first death associated with the use of lignocaine occurred in 1951[11,12]. Toxic reactions had been noted in several of the early clinical trials and further instances were reported by Ansbro *et al.*[13], Gordon[14], Dutton[15], Bennett[16], Bingham and Malherbe[17] and Hartley[18]. Simple overdosage was blamed in all cases. The following new case report is typical.

CASE REPORT

A primigravida, twenty-four years of age, had been in labour for forty-three hours following surgical induction. A total dosage of pethidine 500mg, papaveretum 40mg and scopolamine 0.8mg had been administered

during the first stage of labour. Analgesia during the second stage was provided by the intermittent inhalation of trichlorethylene vapour 0.35 per cent in air.

Forceps delivery was decided upon and bilateral pudendal nerve block was performed using approximately 40ml of plain lignocaine 2 per cent (800mg). Forceps extraction was attempted, but it was found that preliminary manual rotation of the infant's head would be necessary. General anæsthesia was requested and atropine 0.6mg was given intravenously.

Meanwhile, the patient had become restless and twenty-five minutes after the pudendal block had been performed she began to struggle violently and to cry out: 'I'm going to die'. Six minutes later induction of anæsthesia was begun, using nitrous oxide, oxygen and halothane, but after two minutes a severe, generalised, clonic convulsion occurred, lasting for about ten seconds and this was rapidly followed by four others during the next one and a half minutes. Oxygen 100 per cent was given and the convulsions were controlled by the intravenous administration of thiopentone 175mg. Suxamethonium 75mg was given, an endotracheal tube was passed and anæsthesia was continued with nitrous oxide and oxygen and further intermittent doses of suxamethonium.

Forceps extraction of a live infant was accomplished fourteen minutes after the first convulsion and anæsthesia was discontinued twenty-six minutes later. The patient awoke after a further fifteen minutes and her subsequent progress was uneventful.

DISCUSSION

Lignocaine has been used with greater caution following reports of toxic reactions and a reduction in recommended doses and concentrations has been urged by Moore[19], Dutton[15], Bingham and Malherbe[17], Hartley[18] and Walker *et al.*[20]. A maximum permissible adult dose of 500mg is now generally accepted. The trend towards the use of lower concentrations of lignocaine is indicated by the demand for the drug experienced by one of the manufacturers[21]. In 1955 the quantity of the 2 per cent solution sold was more than double that of the 1 per cent solution, but in 1959 the two concentrations were in equal demand.

Routine use of lignocaine 2 per cent continues, however, and this practice greatly increases the risk of toxic reactions. The use of lignocaine in a concentration of over 1 per cent is indicated only for topical application, for epidural and spinal analgesia and in ophthalmic and dental surgery and, in any case, for most of these purposes special preparations of the drug are used. Lignocaine 0.5–1 per cent is suitable for peripheral nerve-block techniques and 0.25–0.5 per cent for infiltration. A total dose of 500mg should not be exceeded in an adult and special care is necessary if the patient is a child. The addition of adrenaline to delay absorption of the drug is valuable.

As with all local analgesic agents, the following precautions should be observed when lignocaine is used: premedication with a barbiturate

if possible; facilities for inflation with oxygen should be to hand; an ultra short acting barbiturate should be immediately available to control convulsions.

SUMMARY

A short review of the introduction of lignocaine into clinical practice is presented and published work relating to the toxicity of the drug is discussed.

A case of toxic reaction to lignocaine is reported.

The routine use of lignocaine 2 per cent is condemned.

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NEW INVENTIONS

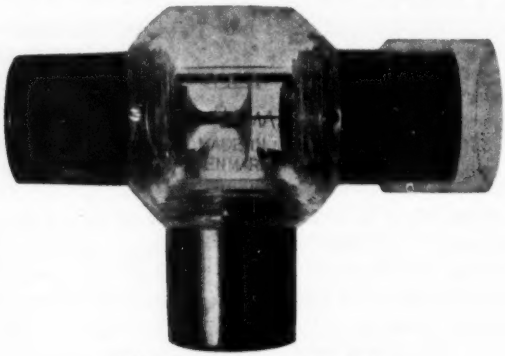
Unless expressly stated by the authors no claim to originality or priority is made and items given under this head include modifications to existing apparatus. The Editor regrets that space considerations make it impossible to publish correspondence on this subject

Attachment for the Ruben valve

C. H. HOSKINS

During six months' experience with the Ruben non-rebreathing valve for anæsthetics where the patient breathed spontaneously, only one disadvantage was found. This occurred during surgery of the head and neck.

Movement of the plunger of the spring-loaded valve disc in the outlet channel can become obstructed, either by drapes or by the hand or arm of the surgeon, or of his assistant. This results in obstruction to the free passage of expired gases. With the continued inflow of gases from the anæsthetic machine and accumulation of expiratory gases in the circuit, sudden release of the obstruction to the valve disc in the outlet channel forces the yarn-spool-like valve across to close the



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outlet channel. As a consequence, the patient is subjected to the increasing pressure of the inflowing gases. This cycle of events can occur surprisingly quickly, particularly with a flow rate of 8 l per minute, and within several seconds the patient's alveolæ and air passages are subjected to quite a high pressure, more so if a cuffed endotracheal tube is used.

On those occasions when this series of events did occur, it was observed in time, but it is conceivable that lung damage could result if it was allowed to persist inadvertently.

To prevent obstruction to movement of the plunger of the valve disc in the outlet channel, the electro-medical technician with the Auckland Hospital Board (New Zealand) made a simple attachment. It consists of a ring of clear perspex 20mm long, with an internal diameter of 23mm tapering to 22mm. This ring fits firmly over the exterior of the outlet channel of the Ruben valve to project about 8–10mm and so protect the plunger of the valve disc (see photograph). The use of this attachment has completely eliminated the described trouble.

The Ruben valve, so modified, was produced by Simonsen and Weel's Eftf. A/S, 43 Bredgade, Copenhagen, Denmark.

Open incubator for neonatal surgery

GORDON J. TAYLOR

The main objective here was to produce an apparatus which would maintain neonatal euthermia during surgery.

It is generally agreed that during the course of an operation the neonate shows a marked tendency to lose heat and to become hypothermic[1]. G. J. Rees[2] remarks that though this is not normally considered to be undesirable in anæsthetised patients, there is in infants a close relationship between hypothermia and the development of sclerema. This disease is more likely to occur in the premature baby.

Various means have been used to prevent the development of hypothermia in anæsthetised neonates, including the use of electric or hot water blankets. The use of these methods has, on occasions, been associated with the occurrence of burns whose importance might be paramount in the case of an ill child. One cause of burning has been

the use of a domestic three-heat electric pad on which the baby has been lying for too long with the pad at its maximum setting. A high pad temperature above the optimum temperature required to maintain the baby in the euthermic state was not considered a disadvantage, provided the baby is well insulated from direct contact with the pad.

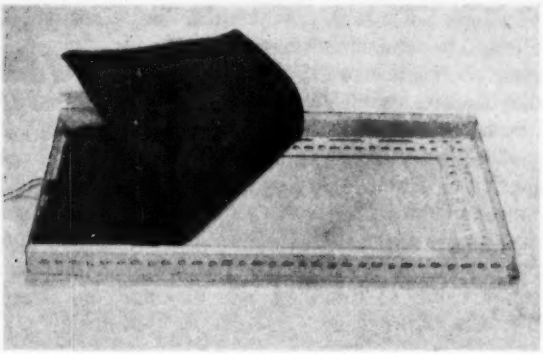

FIG. 1 Incubator frame and base

Thus a temperature gradient would exist from the pad through the insulating layers to the surface of the incubator. Various materials could be used for this layer including air itself, sufficient to drop the pad temperature to an optimum working temperature for the incubator surface on which the baby would lie. This optimum surface temperature had, therefore, to be determined by experiment. The higher pad temperature would ensure that this optimum temperature was reached quickly and with minimal interference from the surroundings. A steady optimum temperature state should, therefore, be the main object in the construction of the incubator.

The apparatus should be of reasonable size, allow of free access to the patient and be both spark and waterproof. Finally it should be easy to assemble from readily available materials at a reasonable cost. The use of a single-heat 150°F electric pad was decided upon with a sealed-in thermostat. The electric lead would connect to a spark-proof power socket without an intervening switch, thus making the whole apparatus spark proof. This pad temperature was chosen as being the most suitable after consulting the manufacturers.

It was decided to enclose the electric pad in a framework of Dexion mounted on block-boarding and covered over with a lid of peg-boarding resting on supports inside the Dexion frame. A hood-like

cover of anti-static rubber sheeting was made to measure to fit over the incubator.

As a result of numerous experiments during various neonatal operations with the apparatus, a satisfactory optimum surface temperature was finally obtained by having the incubator always assembled in the following way: the electric pad was placed in the bottom of the 1½in

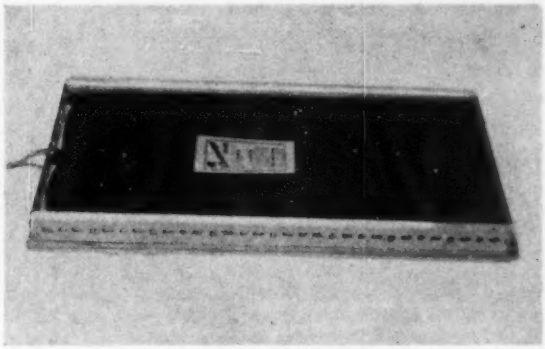

FIG. 2 Electric pad in position

deep Dexion frame covered by the lid of peg-boarding, thus leaving an air space of about ⅝in deep between the pad and the lid. The rubber cover was then fitted and two layers of cellular weave cotton blanket placed on top of the open incubator for the baby to lie on. The surface temperature obtained averaged 100°F, the lowest reading being 98.8°F and the highest 102°F. No red areas of skin were found on any of the babies after operations lasting two-and-a-half to three hours. The temperature readings were taken with a clinical thermometer placed in various positions on the surface of the incubator during use with the usual surgical drapes in position over the baby. Axillary and rectal temperatures were used as appropriate in assessing the temperature of a baby and showed the apparatus to be both safe and practical. No baby was found to have an abnormal temperature which could be attributed to the apparatus. The complete open incubator was assembled as shown in the photographs. It has the same width as the head end of the pædiatric operating table and stands on the anti-static rubber mattress.

To make the rectangular framework, four lengths of 1½in by 2⅜in section Dexion are used. The frame measures 24in by 12in and is held together with four ¼in Dexion bolts. A piece of ⅞in thick block-

boarding 24in by 12in is used for the base and has holes drilled part way through at the four corners correctly sited to recess the projecting bolt heads of the Dexion frame, which can then be screwed flush to the base

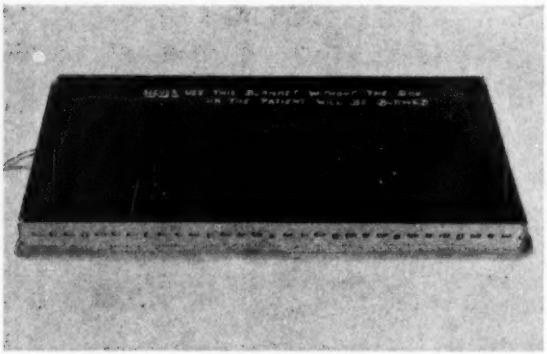

FIG. 3 Incubator lid fitted

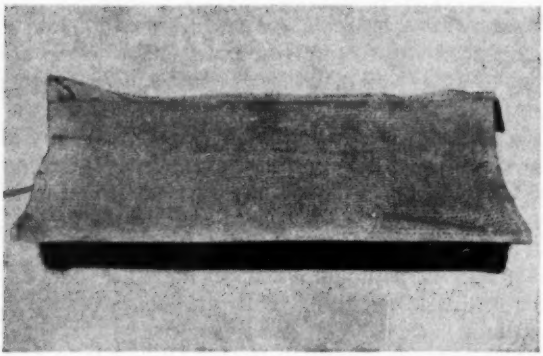

FIG. 4 Open incubator ready for use with its coverings of anti-static rubber and cotton blanket

with short screws. Two wooden laths 1⅛in by ½in and just short of 24in long are made so as to be a driven fit in the positions shown in the photographs where they support the peg-board lid which measures 23⅞in by 11⅞in.

The electric pad supplied by the manufacturer satisfied all the requirements. It measures 23in by 11in and has a rating of 220 volts, 70 watts with a pad temperature of 150°F incorporating glass sealed snap action thermostats. The waterproof electric cabling passes through a rubber grommet in the Dexion frame.

Once assembled the open incubator should be clearly labelled with a warning for the electric pad not to be used apart from the incubator as severe burning from direct contact with the pad would then result. Should soiling occur during use, the rubber cover is easily washed afterwards and the double layer of cotton blanket replaced.

Acknowledgements

I wish to thank Dr H. L. J. Wilson, FFARCS, and Mr R. B. Zachary, FRCS for their kind help and guidance in the preparation of this paper. Thanks are also due to the theatre staff of the Children's Hospital, Sheffield for their ready co-operation and to Mr J. Larway, AIBP for the photographic work. I should like to acknowledge the technical advice and assistance provided by Thermega Ltd, from whom the special electric pad can be obtained.

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Reception-resuscitation cots for the newborn

R. D. LEVIS

The traditional crib and outmoded, almost dangerous oxygen equipment are common in maternity departments. The crib is safe in that the furnishings are sparse, the babe visible and the babe cannot fall out. It allows the administration of oxygen by catheter or funnel and the use of mucus extractors, but no more than this.

The severely asphyxiated babe is the one who has no signs of respiratory effort, or only gives a few feeble gasps after three minutes have

elapsed. Such a case should be accessible for laryngoscopy and endotracheal intubation without waste of time, transferring him to some special resuscitation trolley or table. Handling and undue movement may aggravate shock. Therefore, the best type of resuscitation cot is the one which is also the routine reception cot. It must be simple and relatively inexpensive so as to be provided in every labour room or theatre. It must have the safety of the crib to be acceptable to the nursing staff. The babe's head must be accessible through a drop-end.

FIG. 1 illustrates a perspex cot made at the Central Middlesex Hospital in 1955. The aneroid manometer is calibrated in cm/H_2O, but is breakable. The bag mount and valve are fitted to the wall of the

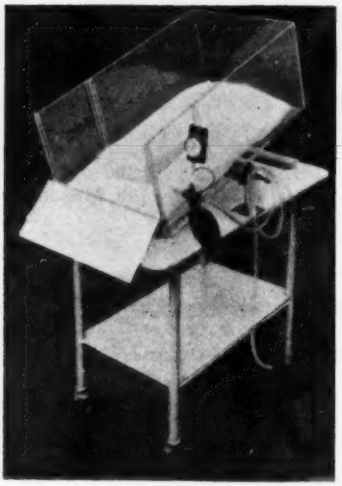

FIG. 1

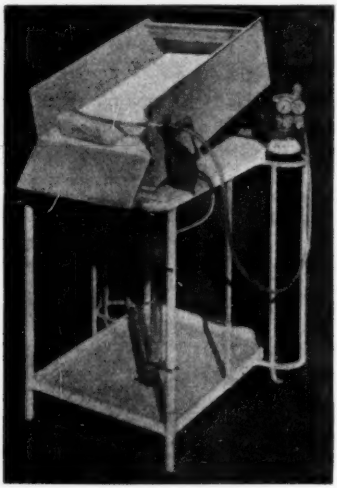

FIG. 2

cot and a nine inch length of tubing lies on the mattress available to connect to funnel, catheter, or endotracheal tube.

FIG. 2 shows a more robust tilting cot with a drop-end fitted with safety catches. It has cradles for two oxygen cylinders, of the same size as those used on the anæsthetic machines. The pin-index yoke, reducing valve and vertical 2 litres flowmeter unit is easily transferred from one cylinder to another. The reservoir bag with expiratory valve is mounted as before, but with an underwater seal instead of an aneroid manometer. This operates as a pressure gauge as well as being a simple safety device. The outer tube is filled with water to 20cm. The tray at the foot

end of the cot is for the laryngoscope, spare batteries and endotracheal tubes. Uncut 00 portex tubes are mounted on tapered glass adaptors with the internal bores of the tapers being equal to that of the tubes. They are sterilised and sealed in paper packets.

In practice, inflation of the lungs is carried out firstly by mouth to glass adaptor in order to be sure the tube is correctly placed and to ascertain whether the trachea is clear or obstructed. If meconium has entered the trachea, suction by mouth to adaptor will safely aspirate it and fill the tube. This necessitates changing the tube and having another ready. It is most important to have several packets of tubes with each cot. By this means more complicated and less safe methods of suction are avoided and time is saved. It must be stressed that intubation of the flaccid edentulous neonate is easy, provided the posture is correct. That is to say, the cot is horizontal and a wedge pillow is placed under the shoulders thus allowing the elongated and relatively large neonatal head to be extended on the neck.

Acknowledgements

My thanks are due to Mr F. Dewar, Instrument Curator, Central Middlesex Hospital, for making the perspex cot and to Oxygenaire Ltd, Duke Street, W1 for the later model in aluminium. Also to Mr A. Booker, Clinical Photographer for the photographs.

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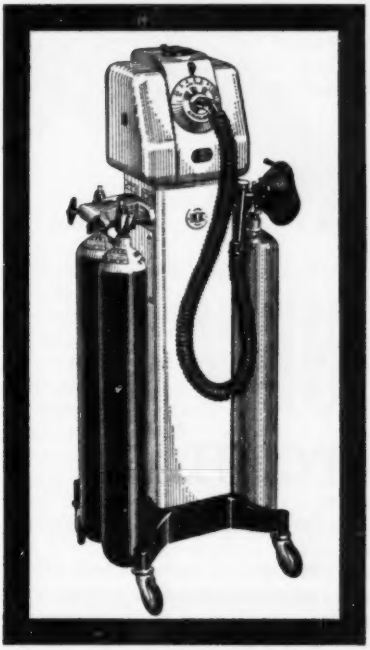
LUCY BALDWIN APPARATUS FOR OBSTETRIC ANALGESIA



The Lucy Baldwin Apparatus is designed for the administration of nitrous oxide and oxygen mixtures for analgesia in obstetric practice. Gas mixture, which may be varied between 70% nitrous oxide/30% oxygen and 100% oxygen, conforms accurately to the value selected on the control dial no matter how draw-off conditions may change. The analgesic properties of nitrous oxide may thus be exploited to the full whilst at the same time maintaining adequate oxygenation. The gas mixture flows only in response to the patient's inspirations and safety cut-off valves eliminate any possibility of inhalation of pure nitrous oxide in the event of undetected failure of the oxygen supply. We shall be glad to send you illustrated literature on request.

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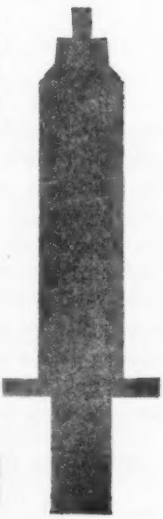
NEW

PENTOTHAL

in disposable rectal syringe

A new dosage form of Pentothal is now available—Rectal Pentothal Suspension—supplied in a disposable syringe. This is a specially designed plastic syringe called the Abbo-Sert which is ready for immediate use. It is provided with two detachable rectal applicators. Rectal Pentothal Suspension in the Abbo-Sert offers significant advantages:

- **No necessity to prepare the drug.**
- **Smaller volume of drug.**
- **No need for bowel preparation.**
- **Obviates the need for buttock strapping.**
- **Absorption uniform.**
- **Onset of action gradual and consistent.**



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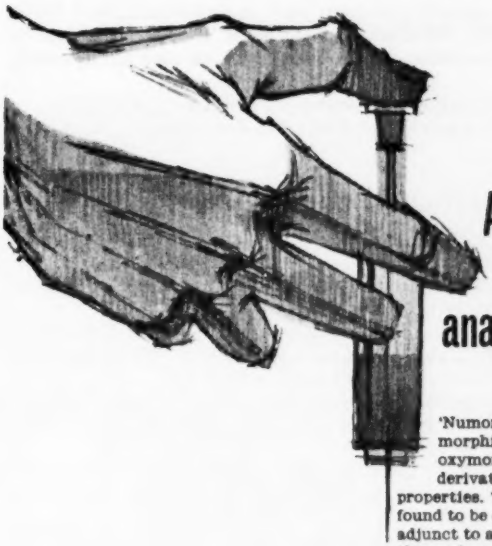

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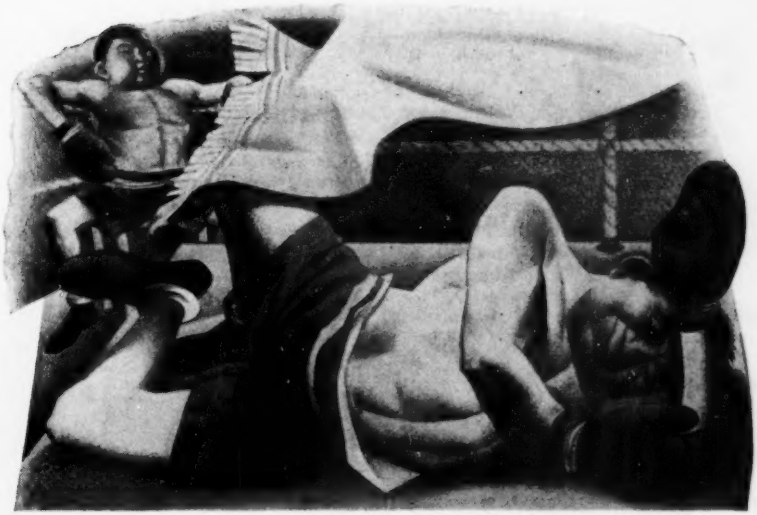

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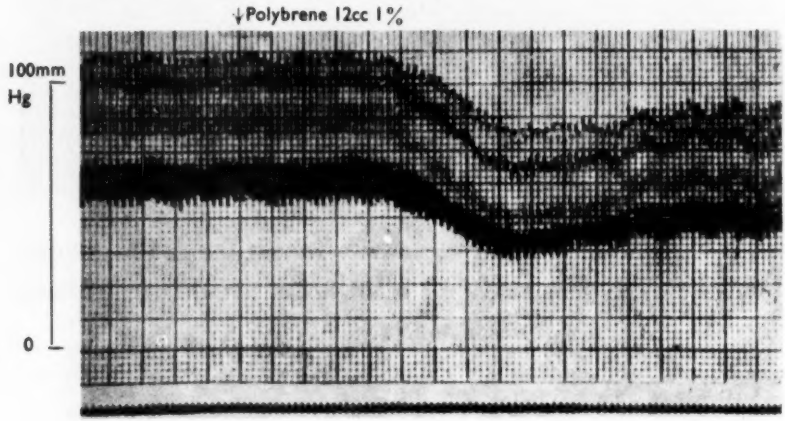
The Editor, *Anæsthesia*

Sir,

The neutralisation of heparin induced anticoagulation by hexademethrine bromide (Polybrene) has been stated to be relatively free from the disadvantages of protamine sulphate[1]. The hypotension that may be caused by the intravenous injection of protamine sulphate has been a serious objection to its use and it was suggested that this could be avoided by the use of hexademethrine bromide.

We have found that significant hypotension may also be produced by the administration of hexademethrine bromide and would agree with Weiss *et al.*[2] that the drug should be administered as a dilute solution and with considerable caution to minimise the severity of the hypotension.



Record of the blood pressure in common iliac artery recorded on Sanborn apparatus, using a NEP pressure transducer, of a patient undergoing hind limb perfusion with cytotoxic drugs.

The illustration shows the arterial blood pressure of a patient to whom hexademethrine bromide was administered to reverse the effects of heparin. In this case the drug (12ml) was administered undiluted into a fast intravenous infusion during the course of three to five minutes.

Yours faithfully,
S. A. Feldman
J. R. Hoyle

References

[1] Polybrene (hexademethrine bromide). (1959) – Communication from Abbott Labs., Chig. Ill. U.S.A.

[2] WEISS, W. H., GILMAN, J. S., CATENACCI, A. J. and OSTERBERG, A. E. (1958). *J.A.M.A.*, *166*, 603.

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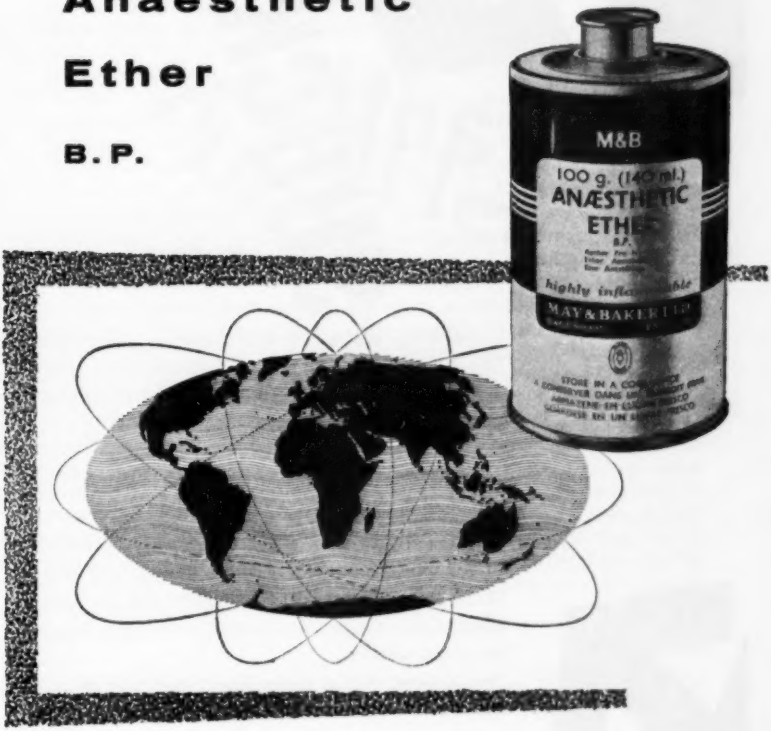

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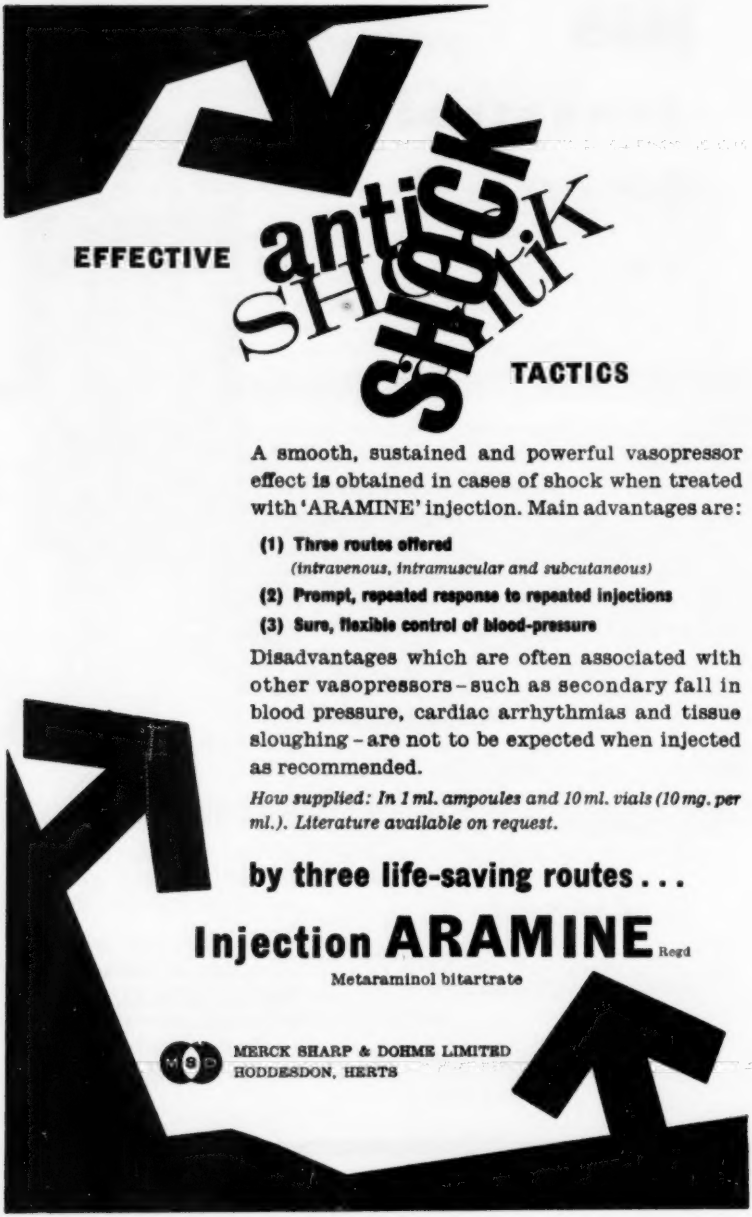

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Obituary

Dr William Stanley Sykes died suddenly on 31st March 1961 at the age of sixty-seven.

Dr Sykes was educated at Rossall, Emmanuel College, Cambridge and at St Bartholomew's Hospital. During the First World War he served in the Navy and held house appointments at Bart's. He then entered general practice at Morley, his birthplace, and took a special interest in anæsthesia, securing appointments at Leeds General Infirmary and at St James' Hospital. During the late war, Dr Sykes served as an anæsthetist in the RAMC with the rank of major and was attached to the 26th General Hospital. He was a prisoner of war in Greece for four years, during which time he did sterling work under very trying conditions. This was recognised by his being appointed MBE. On his return to Leeds, an unfortunate disagreement with the hospital authorities led to his giving up anæsthetics and returning to general practice which he continued until 1959.

Dr Sykes had a real literary gift and wrote several successful thrillers such as 'The Ray of Doom' and 'The Mystery of the Missing Money Lender'. His first medical book 'Manual of General Medical Practice' appeared in 1927 and was followed in 1931 by his work on anæsthetics in the Modern Treatment series. His last work, Volume I of 'Essays on the First Hundred Years of Anæsthesia' was reviewed in '*Anæsthesia*' in July 1960. This book is quite outstanding and reveals the author as one who would go to any lengths to ascertain the truth of a matter and who would then record it faithfully, if sometimes with sarcasm and with no attempt to pander to popular opinion. This uncompromising attitude undoubtedly prevented Dr Sykes from attaining greater heights in his profession, but it secured for him what was of far greater value, the respect of his colleagues for his absolute integrity. It is to be hoped that work on subsequent volumes is sufficiently advanced to make publication possible.

Dr Sykes leaves a widow and stepdaughter to whom we offer our sincere sympathy.

Association News

Research Prizes 1961. The Council of the Association has pleasure in announcing that the following awards have been made: Dr J. D. Robertson and Dr A. W. Williams for their paper on 'Studies on the clinical and pharmacological effects of hydroxydione': Dr A. C. Forrester and Dr J. Brown for their paper on 'The prediction of œsophageal temperature in hypothermia' which has already been published in '*Anæsthesia*': Mr D. G. Calvert for his paper on 'Inadvertent hypothermia in pædiatric surgery and a method for its prevention'.

The Council of the Association regrets that, on the advice of its assessors, no prize can be awarded for an essay dealing with **dental anæsthesia.** In recognition of the industry displayed, however, it has been decided to award twenty guineas each to four of the entrants on an *ex gratia* basis.

A Scientific Meeting for Registrars, open to all junior anæsthetists up to and including those of senior registrar status, whether or not they are members of the Association, will be held in Manchester on Friday, 30th March 1962. Further details will be available later.

The Annual General Meeting of the **Scottish Society of Anæsthetists** was held at Gleneagles Hotel, Perthshire, from 28th to 30th April 1961.

The following office bearers were elected:

PRESIDENT	Dr J. W. L. Bain, *Aberdeen*
VICE-PRESIDENT	Dr Margaret Muir, *Dundee*
HON. SECRETARY	Dr M. Shaw, *Victoria Infirmary, Glasgow*
REGIONAL REPRESENTATIVES	Dr James Crawford, *Glasgow* Dr A. C. Milne, *Edinburgh* Dr T. R. MacDonald, *Dundee*

The Presidential address was 'An exciting twenty years'. The Guest Speaker was Dr G. S. W. Organe who spoke on 'Atlantic Curtain'. The Registrar's Prize was won by a paper submitted jointly by Dr D. D. Moir and Dr J. S. Reid of Stobhill Hospital, Glasgow on 'Post-operative respiratory difficulties'.

The spring meeting of the **Anæsthetists of the North East Metropolitan Region** was held at the London Hospital on Saturday, 27th May 1961. Short papers were read on a new ventilator, the use of urea in neurosurgery, closed-circuit halothane, the recovery room, the artificial kidney, ion exchange resins, and the dry field in orthopædic surgery. The showing of three films concluded a most successful meeting.

Faculty News

Burroughs Wellcome Fellowships. The Burroughs Wellcome Fund in the USA has founded a Fellowship in Anæsthesia at the University of California Medical Centre, Los Angeles, to give a British physician in training in anæsthetics the opportunity of the cultural, academic and professional experience of an American university and to increase the understanding of anæsthetics between the physicians of the United States and Great Britain. The value of the Fellowship is 4,000 dollars p.a., including travelling expenses. This amount has been found by the first Fellow to be adequate to cover normal requirements. The Fellowship has been founded to support Fellows for one year each and the Fellows are expected to return to Britain upon the completion of the terms of their respective Fellowships. The Fellowship is for both clinical and research work.

Anæsthetists who have passed at least the Primary Examination for the FFARCS are invited to apply for the third annual award. The Fellow appointed will be expected to take up his duties at the first convenient date after 1st September 1961. Applications, giving details of qualifications and experience (including research experience, if any) and the names of two persons to whom reference may be made if necessary, should be sent to R. S. Johnson-Gilbert, MA, Secretary, Faculty of Anæsthetists, Royal College of Surgeons of England, Lincoln's Inn Fields, London WC2.

Election of Dean and Vice-Dean. At the last meeting of the Board of Faculty, Professor W. W. Mushin was elected Dean for the coming year and Dr H. J. V. Morton was re-elected Vice-Dean.

Election to the Fellowship in the Faculty. Under the provision for the election of five Fellows in the Faculty annually, the Council of the College, on the recommendation of the Board of Faculty, has made the following elections: Professor M. S. el Hakeem, *Cairo*; Dr C. S. Jones, *Cape Town*; Dr L. H. Lerman, *London*; Dr O. I. E. Nicholson, *Manchester*; Dr V. Rajagopalan, *Madras*.

Annual Meeting and Thirteenth Anniversary Dinner. The Annual General Meeting was held on 15th March and was followed by the Frederic Hewitt Lecture given by Dr R. P. W. Shackleton, and in the evening the Thirteenth Anniversary Dinner was held. It was attended by 238 people.

Scientific Meeting. The Faculty had a very successful Scientific Meeting at the College on 6th May, which was attended by over 400 people. The subject was 'Hypothermia'.

Faculty of Anaesthetists, Royal College of Surgeons in Ireland

The first examination for the Primary Fellowship of the Faculty of Anæsthetists of the Royal College of Surgeons in Ireland will commence on 13th November 1961; entries must be lodged with the Registrar, RCSI, St Stephen's Green, Dublin 2, twenty-one days beforehand.

Commonwealth and Foreign News

The First European Congress of Anæsthesiology will be organised by the Austrian Society of Anæsthesiology in Vienna from 3rd to 7th September 1962.

The former Imperial Palace will serve as the meeting place, where there will be a Scientific Congress as well as a Postgraduate Medical Training Course.

English, French and German will be the working languages. Provisions will be made for simultaneous interpretation.

The main subjects of the Scientific Congress are: watch stations and special care units; old patients and anæsthesia; anæsthesia problems in acute surgery (accident problems excluded); free papers (ten to fifteen minutes) in limited numbers.

This programme will be supplemented by a series of symposia on specific problems of anæsthesia and related questions. Participants who wish to organise a symposium (about ninety to one hundred and twenty minutes) should contact the Scientific Secretary – who will extend also any other information in relation with the scientific programme – by 30th September 1961, at the very latest. These participants should indicate, at the same time, the subject of their symposium as well as the names of the intended discussants. (Address: Dr Karl Steinbereithner, Postgraduate Medical School, 4 Alserstrasse, Vienna IX.)

A preliminary programme will be circulated in the autumn of 1961. It will include registration forms. For information apply to the Secretary General, Dr Rudolf Kucher, Wien, Medizinische Akademie IX, Alserstrasse 4. Tel. 33 94 19.

The eighth **Brazilian Congress of Anæsthesiology** will be held from 15th to 21st October 1961 at Goiânia. Enquiries should be addressed to Post Box 245, Department of Anæsthesia, Medical Association of Golas, Goiânia, Brazil.

A congress of the **Societies of German, Austrian and Swiss Anæsthetists** will be held in Geneva from 8th to 10th September 1961. The main subject will be the control of bleeding during operations under general anæsthesia. The introduction will be by Sir Robert Macintosh.

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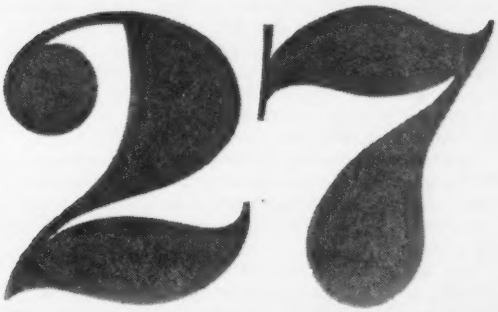

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RESEARCH PRIZES · 1962

The Association of Anæsthetists of Great Britain and Ireland is offering three prizes annually (of £100, £50 and £25) for papers based on research in anæsthesia or related subjects. Entrants need not be members of the Association, but the work described must have been carried out in Great Britain, Ireland or the British Commonwealth. Papers shall not exceed 5,000 words in length and shall be typewritten in English. Three copies of the essay shall be submitted.

Papers must be original, and refer to work which has not previously been published, though an exception to this rule will be made in the case of papers which have been published in the journal '*Anæsthesia*' during the preceding year. Prize winning entries will become the property of the Association.

The decision of the Council of the Association of Anæsthetists in all matters relating to the research prizes shall be final. Essays shall be submitted to the Honorary Secretary of the Association of Anæsthetists of Great Britain and Ireland, 47 Lincoln's Inn Fields, London WC2, on or before 30th March 1962.

RESEARCH TRUST FUNDS

The Council of the Association will be pleased to consider applications from Fellows and Members needing financial assistance for educational or research purposes. Applicants for travel grants should state the objects of their proposed visits and submit detailed itineraries. Applicants for grants for research should state not only the objects of their investigations but also their proposed methods of experimentation. In all cases applicants should provide evidence that they have been unable to procure any or all of the necessary assistance from local sources. Any apparatus acquired through this fund remains the property of the Association and must be made available for the use of other Fellows or Members if subsequently required.

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